A STUDY OF THE EFFICACY OF TRAMADOL AS AN ADJUVANT TO BUPIVACAINE IN BRACHIAL PLEXUS BLOCK

Dissertation submitted for the degree of
DOCTOR OF MEDICINE
Branch – X (ANAESTHESIOLOGY)

APRIL – 2013

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY,
CHENNAI, TAMIL NADU.

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF THE EFFICACY OF TRAMADOL AS AN ADJUVANT TO BUPIVACAINE IN BRACHIAL PLEXUS BLOCK"** is a bonafide record of the work done by **Dr. HARIBASKAR R** under my supervision and guidance in the Department of Anaesthesiology at Thanjavur Medical College, Thanjavur during the period of his post graduate study from April 2010 to March 2013 for the partial fulfillment of M.D. (Branch X - Anaesthesiology) degree.

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DECLARATION

I, solemnly declare that the dissertation titled **"A STUDY OF THE EFFICACY OF TRAMADOL AS AN ADJUVANT TO BUPIVACAINE IN BRACHIAL PLEXUS BLOCK"** is a bonafide work done by me at Thanjavur Medical College Hospital, Thanjavur, during 2010 – 2013.

The dissertation is submitted to **"The Tamilnadu Dr. M.G.R. Medical University, Chennai",** Tamilnadu as a partial fulfillment for the requirement of **M.D** Degree examinations– Branch -X (Anaesthesiology) to be held in April 2013.This has not been submitted previously by me for the award of any degree or diploma from any other university.

Place: Thanjavur
Date:

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CONTENTS

ABSTRACT

Background and objectives: Supraclavicular plexus block provides good alternative to General anaesthesia for upper limb surgeries with good postoperative analgesia. Various drugs have tried as adjuncts to local anaesthetics for brachial plexus block to enhance the quality and duration of analgesia. The present study was undertaken to assess the effect of Tramadol added to brachial plexus block by supraclavicular approach for onset and duration of block and postoperative analgesia.

Methods: A prospective, randomized, double blinded study was conducted on 60 ASA I or II adult patients undergoing upper limb surgeries under supraclavicular brachial plexus block. Patients were randomly divided into two groups. Patients in Group B (n = 30) were administered 38mL of 0.25% Bupivacaine + 2ml Normal saline and Group BT (n = 30) were given 38mL of 0.25% Bupivacaine + 2ml Tramadol (2mg/kg). The onset time and duration of sensory and motor blockade were recorded. Haemodynamic variables (i.e., heart rate, systolic and diastolic blood pressure, oxygen saturation), and rescue analgesic requirements were recorded for 24 hrs postoperatively.

Results: The onset of sensory and motor block was significantly faster in Group BT compared to Group B ($P < 0.05$). Rescue analgesic requirements were significantly less in Group BT compared to Group B ($P < 0.05$). Haemodynamic variables did not differ between groups in the post-operative period.

Conclusion: Thus Tramadol (2mg/kg) in combination with 38mL of Bupivacaine (0.25%) was found to be good agent for hastening the onset of sensory and motor block and improved postoperative analgesia when used in brachial plexus block without producing any adverse events.

Keywords: Supraclavicular brachial plexus block; Tramadol.

1.INTRODUCTION

"Man uses his arms and hand constantly… as a result he exposes his arms and hands to injury constantly… Man also eats constantly… Man's stomach is never really empty… The combination of man's prehensibility and his unflagging appetite keeps a steady flow of patients with injured upper extremities and full stomachs streaming into hospital emergency rooms. This is why the brachial plexus is so frequently the anaesthesiologist's favorite group of nerves" - Classical Anaesthesia Files, David little, 1963[1].

Brachial plexus block is an alternative technique to general anaesthesia for upper limb surgeries. They produce complete muscular relaxation, maintaining stable intraoperative hemodynamic condition and sympathetic block which reduces postoperative pain.

Brachial plexus block is used today to provide anaesthesia for upper limb surgeries. There are four usual sites of approach.

1. Interscalene approach
2. Supraclavicular approach
    a. Classic approach
    b. Plumb –bob technique
    c. Subclavian perivascular technique

3. Axillary approach

4. Infraclavicular approach

Among the four approaches, Supraclavicular brachial plexus block is a very popular mode of anaesthesia for various upper limb surgeries. This approach is attractive due to its effectiveness in terms of cost and performance, margin of safety, along with good postoperative analgesia. It also has the reputation of providing most complete and reliable anaesthesia for upper limb surgeries. The plexus is blocked at the level of trunk where it is most compact i.e. at the middle of brachial plexus, resulting in homogenous spread of anaesthetic throughout the plexus with a faster onset and complete block.

Bupivacaine is one of the commonly used local anaesthetics as it has a longer duration of action varying from 3 to 8 hours. However, it has limiting factors like delayed onset, patchy or incomplete analgesia. To minimize these drawbacks many drugs like neostigmine, opioids, hyaluronidase, midazolam, clonidine etc., have been added to local anaesthetics to improve the quality and duration of action and postoperative analgesia.

A variety of opioids have been studied for brachial plexus blockade including tramadolκ. Tramadol is a synthetic 4-phenyl-piperidine analog of codeine has a unique mode of action. First, it stimulates the μ receptor and to lesser extent δ and κ-opioids receptors. Then by nonopioid mechanism it also activates spinal inhibition of pain by decreasing the reuptake of norepinephrine and serotonin from the nerve endings and potentiates the effect of local anaesthetics when mixed together in peripheral regional nerve block. It has less respiratory depressant effect due to weak μ receptor affinity.

The present study is being undertaken to evaluate the onset time, duration and postoperative analgesic efficacy of bupivacaine and tramadol for brachial plexus block by supraclavicular approach.

1. AIM OF THE STUDY

To evaluate the effects of adding tramadol (2mg/kg) as an adjuvant to bupivacaine (0.25%) in brachial plexus block by supraclavicular approach with regard to the following parameters:

- Onset time and duration of sensory blockade
- Onset time and duration of motor blockade
- Duration of analgesia
- Untoward side effects
- Hemodynamic variables
- Number of rescue analgesics in the postoperative 24hours

2. HISTORY

HISTORICAL REVIEW

"History, although sometimes made up of the few acts of the great, is more often shaped by the many acts of the small"

- Mark Yost.

1901- Harry Cushing's first used the term Regional anaesthesia to describe pain relief by nerve block. The term regional analgesia denotes the interruption of pain impulses by physiological blockade at a certain point along their pathway of transmission in the peripheral nerves.

HISTORY OF BRACHIAL PLEXUS BLOCK

1. 1885 - William Stewart Halsted performed the first brachial plexus block.
2. 1886 - Carl Koller demonstrated the anaesthetic properties of cocaine on the eye of patient.
3. 1897 - George Crile used a similar technique in which the plexus was exposed under local anaesthesia in a 12 year old boy.

EVOLUTION OF SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK[2]

1. 1911-1912 - Kulenkampff described the first percutaneous supraclavicular approach. He pointed out that above the clavicle the plexus lies under the skin as it passes over the first rib and accessible to a percutaneous technique.
2. 1922- Labat G advocated an injection at three separate points which failed to elicit parasthesia by Kulenkampff's method. First injection, beneath the deep fascia in the direction of the first rib, second towards the chassaignac's tubercle and third towards the lateral margin of the first rib behind the clavicle (5 ml with each injection).
3. 1926 - Livingston carried out Kulenkampff's technique without the production of parasthesia as soon as the deep cervical fascia had been penetrated. He wrote that the plexus and the artery are separated from the surrounding structures by a fascial investment.
4. 1940 - Patrick chooses to lay down a "wall of anaesthetic" through which the plexus must pass in its course over the first rib, where 60-70 ml of solution was being injected during 5-6 insertions. This technique became the "standard technique" of

supraclavicular block, subsequently referred to by many as the "classical supraclavicular technique".

5. 1942 - Knight modified Patrick's technique by making the three injections through three separate needle insertions, parallel to one another. For the first time he directed the needle insertion caudally.
6. 1944- Murphey used a single injection technique and used lateral border of anterior scalene muscle as the landmark and direction of needle insertion caudal as with Knight's technique, not medial or dorsal, as with most other techniques.
7. 1949 - Bonica and Moore utilized Kulenkampff's and Patrick's technique and developed a technique where it begins with utilizing the classical landmarks and direction of needle insertion and demands a definite parasthesia prior to first injection. Then continued as Patrick's technique by laying down a wall of anaesthetic solution by "Walking the rib" and makes multiple injections during each withdrawal of the needle. This was used over the subsequent twenty years.
8. 1958 - Lookman fully realized the potential of the fascial sheath, who like Livingston realised on the fascial investment of the plexus. He carefully dissected the plexus and said that

plexus lies in a closed compartment. He said this space lies between the anterior and middle scalene muscles and is pyramidal in shape, with its apex pointing upwards and medially towards the extent of the fourth cervical vertebra. He did not verify the needle's proper placement within space before the injection. He admitted the tendency for the point of the needle to pass too posteriorly and hence to come to be within the substance of (or even behind) the middle scalene muscle.

9. 1964 - Winnie after numerous anatomical dissections showed that the relation of the plexus and the subclavian artery to the midpoint of the first rib is not constant. He showed that there is a constant relationship between the anterior and middle scalene muscles, the plexus and the first rib. The plexus between the scalene muscles always insert on the first rib. He inserted needle between the two muscles in the direction of the space between them. Once a parasthesia is obtained, a single injection is made into the space.
10. Fortin and Tremblay advocated the use of a short needle which was long enough to reach the plexus but too short to reach the lung, in an attempt to minimize the threat of pneumothorax.

History of Local Anaesthesia - Bupivacaine

1. 1956 – Bupivacaine was synthesized by Ekenstam.
2. 1963 – Bupivacaine was introduced into clinical practice by Telivuo.

History of Tramadol

1. 1970 - Tramadol was introduced by Grunenthal in German market.

History of Peripheral Nerve Stimulator

1. 1912 Perthes and 1955 Pearson - demonstrated peripheral nerve could be identified by electro stimulation.
2. 1962 Greenblatt and Denson -introduced the nerve stimulator into clinical practice of anaesthesiology.

4. ANATOMICAL CONSIDERATIONS[3]

THE BRACHIAL PLEXUS:

Knowledge of the formation of brachial plexus and of its distribution is absolutely essential for the use of brachial plexus anaesthesia for the upper limb surgeries. Close familiarity with the vascular, muscular and fascial relationships of the plexus throughout its formation and distribution is also essential to the mastery of the various techniques of brachial plexus blockade.

In its course from intervertebral foramina to the upper arm, the fibres form roots, trunks, divisions, cords and terminal nerves passes through a complex process of combining, dividing, recombining and finally redividing.

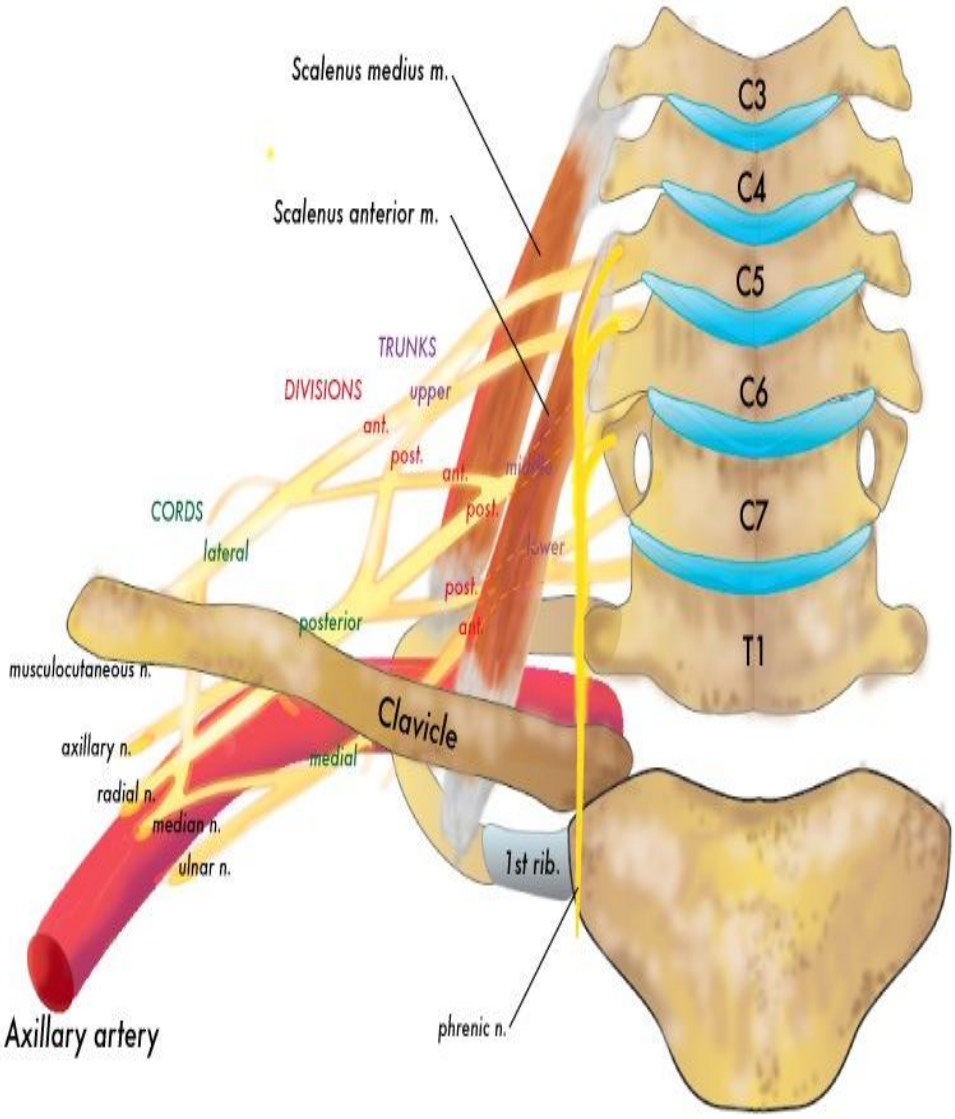
FORMATION OF PLEXUS

Roots

The plexus is formed by the anterior primary rami of the cervical nerves 5^{th} to 8^{th}, together with the bulk of the thoracic nerve 1^{st} (C_8 and T_1). In addition there is frequently a contribution above from C_4 to the 5^{th} cervical root and another below fromT_2 to the 1^{st}

FIGURE 1

THE RELATIONS OF THE BRACHIAL PLEXUS



thoracic nerve. Occasionally the plexus is mainly derived from $C_{4\text{-}8}$ (Pre –fixed plexus) or from $C_6 – T_2$ (post – fixed plexus).

Trunks and Divisions

The five roots of the plexus emerge from the intervertebral foramina. They lie in the gutter between the anterior and posterior tubercles of the corresponding transverse process. All five roots then become sandwiched between scalenus anterior and medius. Here the roots of C_5 and C_6 unite to form the upper trunk. The root of C_7 continues as the middle trunk and those of C_8 and T_1 form the lower trunk. Each trunk then divides behind the clavicle, into anterior and posterior divisions, which unite to form the cords in the axilla.

Cords:

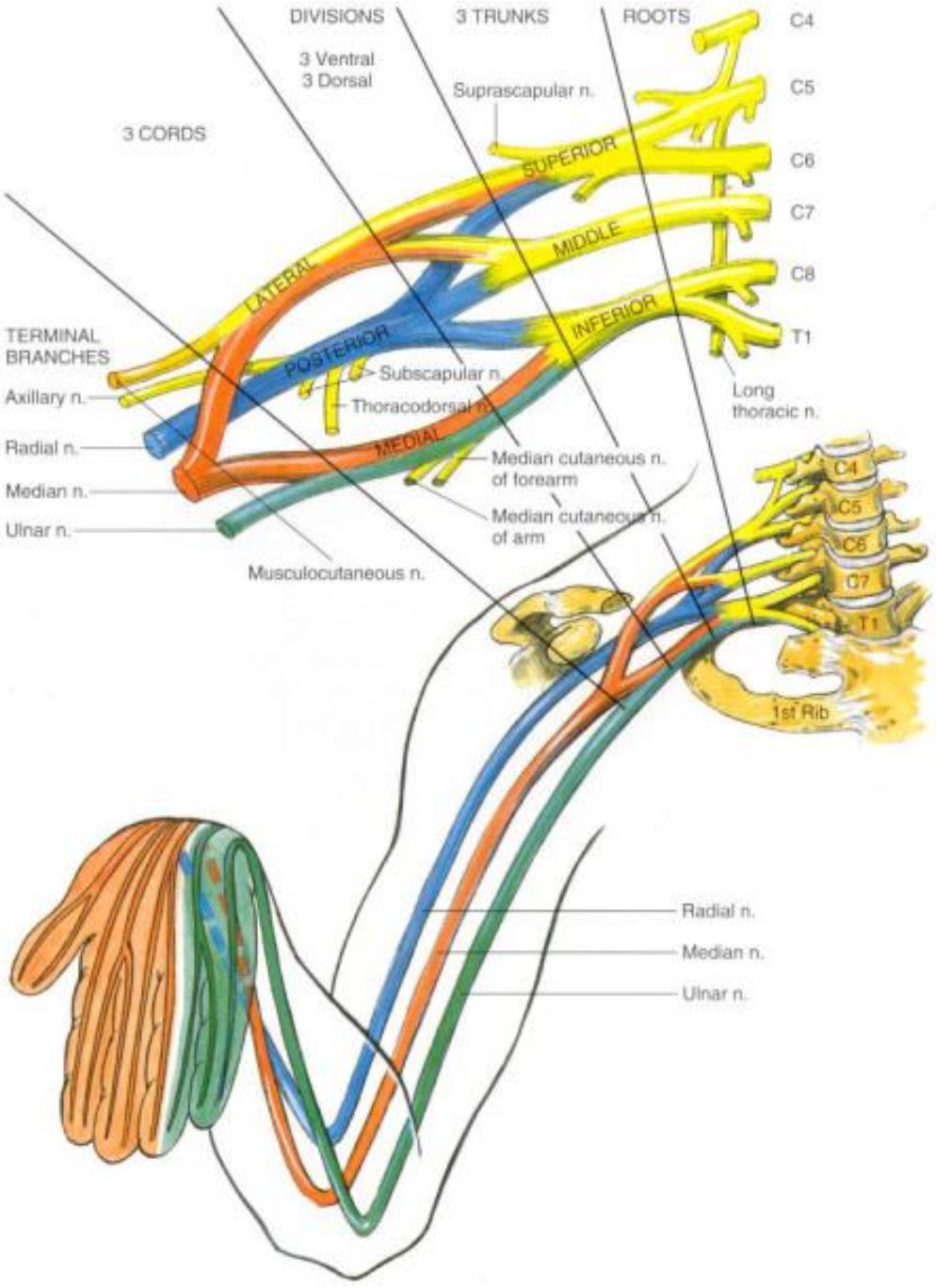
The six divisions unite up into three cords lateral, medial and posterior into the axilla.

These cords are composed as follows:

The lateral cord is formed by the union of the anterior divisions of the upper and middle trunks. The anterior division of the lower trunk forms the medial cord. All the three posterior divisions form the posterior cord.

FIGURE 2

BRANCHES OF BRACHIAL PLEXUS



DISTRIBUTION OF BRACHIAL PLEXUS

These are divided with relation to clavicle - the supraclavicular branches that arise above the clavicle and the infraclavicular branches that arise below it.

The composition of the brachial plexus can be summarized as follows:

SUPRACLAVICULAR BRANCHES

1. Five roots – the anterior primary rami of C_{5-8} and T_1
2. Three trunks.

   Upper trunk, C_5 and C_6

   Middle trunk, C_7 alone and

   Lower trunk, C_8 and T_1
3. Six divisions – each trunk divides into an anterior and posterior division.

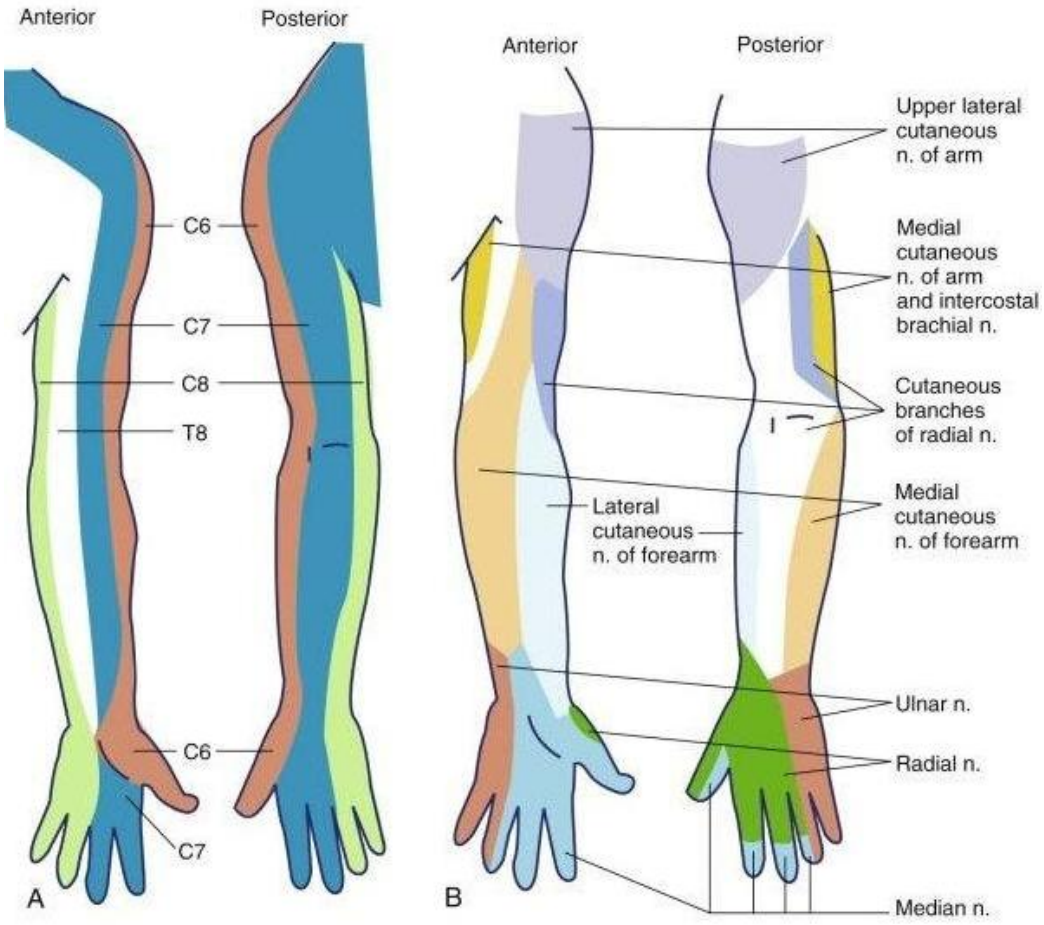
INFRACLAVICULAR BRANCHES

4. Three cords
   a) Lateral cord (C_5 - C_7)
   b) Medial cord (C_8 - T_1)
   c) Posterior cord (C_5 - T_1)

FIGURE 3

A. CUTANEOUS DISTRIBUTION OF THE CERVICAL ROOTS.

B. CUTANEOUS DISTRIBUTION OF THE PERIPHERAL NERVES



Branches are given off from

1. Roots
2. Trunks and
3. Cords

Branches from the Roots

1. Nerve to the serratus anterior (C5, C6 and C7)
2. Muscular branches to
    - i. Longus cervices (C5- C8)
    - ii. Three scalene (C5 – C8)
    - iii. Rhomboids (C5)
3. A twig of phrenic nerve (C5)

Branches from the trunks

1. Suprascapular nerve (C5, C6)
2. Nerve to subclavius (C5, C6)

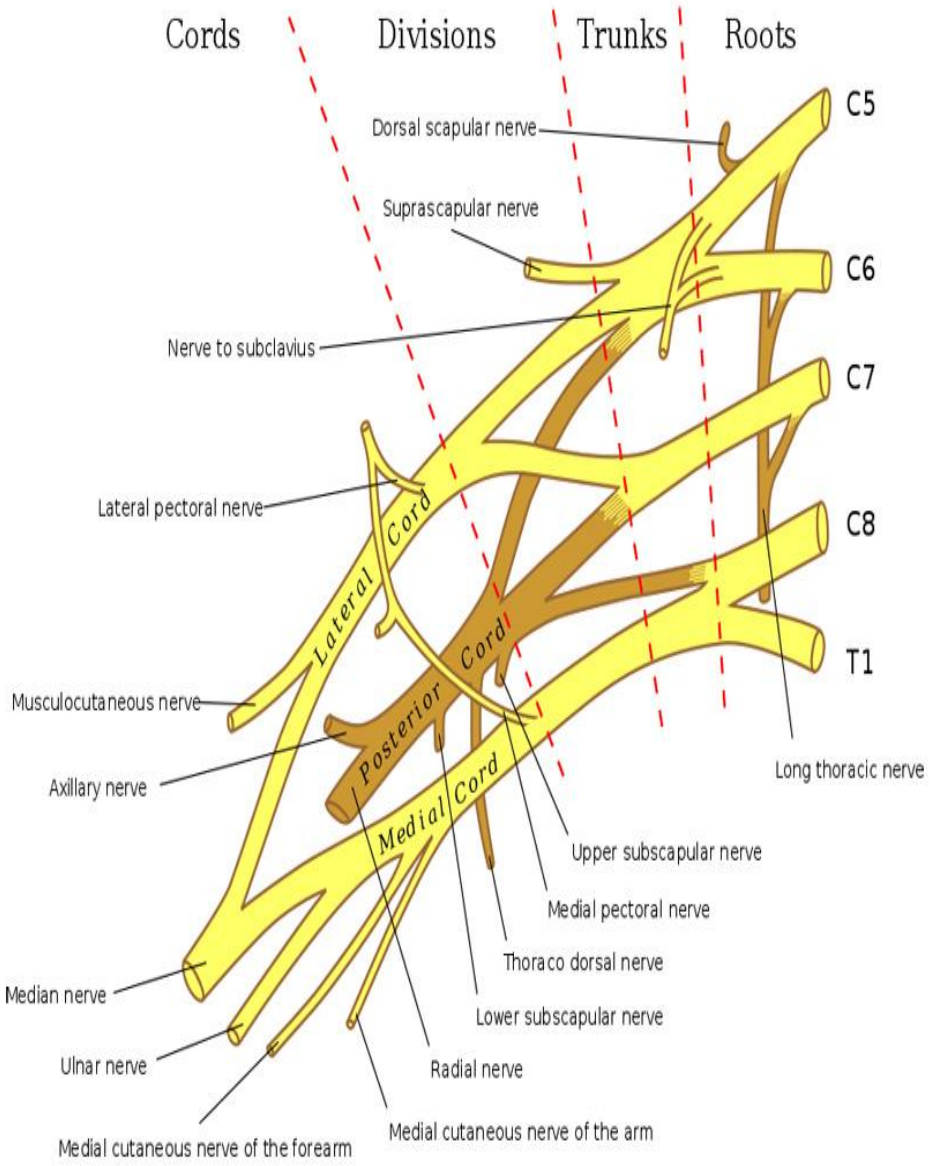
BRANCHES FROM THE CORDS

LATERAL CORD

Lateral pectoral nerve	(C5, C6, C7)
Lateral head of median nerve	(C5, C6, C7)
Musculocutaneous nerve	(C5, C6, C7)

FIGURE 4

FORMATION OF BRACHIAL PLEXUS



MEDIAL CORD

Medial pectoral nerve	(C8, T1)
Medial head of median nerve	(C8, T1)
Medial cutaneous nerve of arm	(C8, T1)
Medial Cutaneous nerve of forearm	(C8, T1)
Ulnar nerve	(C7, C8, T1)

POSTERIOR CORD

Upper subscapular nerve	(C5, C6)
Lower subscapular nerve	(C5, C6)
Nerve to latissimus dorsi	(C6, C7, C8)
Axillary nerve	(C5, C6)
Radial nerve	(C5, C6, C7, C8, T1)

SYMPATHETIC CONTRIBUTION TO BRACHIAL PLEXUS:

The segmental preganglionic sympathetic contributions are variable, but generally extend more caudal. The highest contribution is usually from T2 with T1 contributing only rarely, while lowest may be as far as T8, T9 or even T10.The post ganglionic contributions are from grey rami communicantes from the sympathetic chain.

RELATIONS OF BRACHIAL PLEXUS:

In its passage from the cervical transverse processes to the first rib, the plexus is "sandwiched" between the anterior and middle scalene muscles and invested in the fascia of those two muscles.

The 'interfascial compartment', along with subclavian artery which crosses the first rib immediately infront of the trunks. Artery is close to the scalenus anterior and the plexus is close to the scalenus medius. Subclavian vein is separated from the artery by the scalenus anterior. The fascia covering the muscles is derived from the perivertebral fascia, which splits to invest these muscles and rejoins again at their lateral margins to form an enclosed space, the interscalene space. As the plexus cross the first rib, the three trunks are arranged one on top of the other vertically. Not infrequently, the inferior trunk gets trapped behind and even beneath the subclavian artery above the rib, during embryologic development.

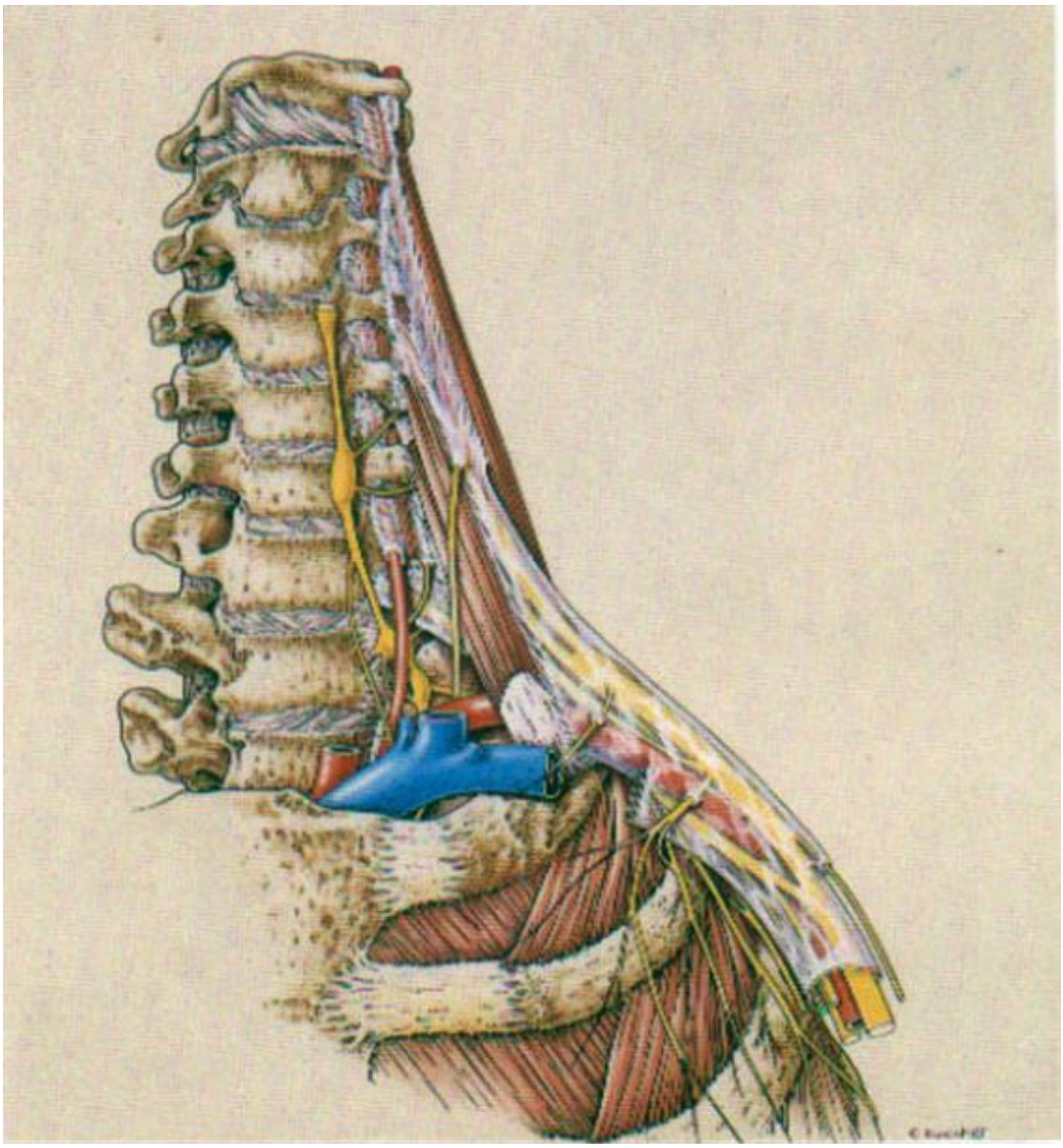
This may be the reason why local anaesthetics injected via the interscalene techniques sometimes fail to provide anaesthesia in the distribution of the ulnar nerve, which may be buried deep within inferior trunk behind or beneath the subclavian artery. After crossing the first rib, they the split to form divisions and the cords and

subclavian artery becomes the axillary artery. Above the clavicle, the axillary artery lies central to the three cords, in the axilla the lateral and posterior cords are lateral to the first part of the axillary artery, the medial cord being behind it. Around the second part of the artery, they are related according to their names. In the lower axilla, cords divide into nerves for the upper limb.

In passing over the first rib under the clavicle, the subclavian vein also becomes the axillary vein and its relationship with the neurovascular bundle changes. Above the first rib the subclavian vein does not lie within the neurovascular bundle, it is separated by the insertion of scalenus anterior. As it passes over the first rib, becoming the axillary vein it joins the neurovascular bundle so that parts of the plexus are sandwiched between artery and vein. As all the three enter the axilla, they invaginate the perivertebral fascia at the lateral margins of the anterior and medial scalene muscles, carrying this fascial investment of the neurovascular bundle into the axilla as the axillary fascia, an extension of the perivertebral or scalene fascia forming the axillary perivascular space, a tubular extension of the interscalene space. In its course through the axilla and upper arm the fascia of the surrounding muscles contribute to the axillary sheath, making it thick and tough, providing the 'fascial

FIGURE 5

SHEATH AROUND THE PLEXUS



click' to the anaesthetic while entering the sheath. It is important to note that major terminal nerves leave the sheath high in the axilla under the cover of pectoralis minor muscle.

The musculocutaneous nerve enters the substance of coracobrachialis and continues down within this muscle. The axillary nerve also leaves the sheath immediately after arising from the posterior cord. The interocostobrachial nerve travels parallel to but outside the axillary sheath and medial cutaneous nerve of the arm runs similarly but occasionally it may remain within the sheath.

THE BRACHIAL PLEXUS SHEATH

The connective tissue of the prevertebral fascia and the anterior and middle scalenus envelops the brachial plexus as well as the subclavian and axillary artery in a neurovascular "sheath".

Volume of the sheath: 42ml.

Shape of the sheath: Cylindrical to conical - Wide proximally and narrow distally.

Length: 8-10cms.

The tissue is densely organized as it leaves the deep cervical fascia proximally, but becomes more loosely arranged distally. The

sheath blends with the fascia of the biceps and brachialis muscle distally.

Anaesthetic implications

Because of these connective tissue septae, anaesthesia might be complete and rapid in onset in some nerves, but delayed and incomplete or completely absent in others. The incidence of partial block is an exception rather than the rule, so septa apparently are of little clinical significance as the local anaesthetic can percolate through them.

TECHNIQUE OF BRACHIAL PLEXUS BLOCK

Surgical anaesthesia of the upper extremity and shoulder can be obtained following neural blockade of the brachial plexus at several sites. The various approaches that can be used for this blockade are as follows.

1. Interscalene approach
2. Supraclavicular approach
    a. Classic approach
    b. Plumb –bob technique
    c. Subclavian perivascular technique
3. Axillary approach
4. Infraclavicular approach.

TECHNIQUE OF BLOCKADE - SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH TO BRACHIAL PLEXUS

Anatomical Land marks:

The three trunks are clustered vertically over the first rib cephaloposterior to the subclavian artery. Neurovascular bundle lies inferior to the clavicle at above its mid point.

The essential landmarks to be identified are

1. Cricoid cartilage
2. Interscalene groove
3. Clavicle midpoint
4. Subclavian artery

PROCEDURE:

Position: Supine position with the head turned to the opposite side to be blocked. The arm is pushed down to depress the clavicle.

The posterior border of sternocleidomastoid is felt, by asking the patient to raise the head while keeping the head turned to opposite side. The interscalene groove should be located behind the midpoint of the posterior border of the muscle. The anterior and middle scalene muscles can be made prominent by asking the patient to inspire vigorously. Approximately 1 cm above the midpoint of the

clavicle the pulsation of the subclavian artery can be felt in the interscalene groove while standing on the side of the patient. On the right side interscalene groove is palpated with the left index finger and the needle is inserted with the right hand. Subclavian artery is guarded with thumb. After aseptic measures and intradermal weal raised with local infiltrations of 1 ml of 2% Lignocaine intradermally in the interscalene groove 1 to 1.5 cm above the clavicle, a 22G, 50 mm short bevelled unipolar insulated needle connected to a nerve locator is directed posteriorly and caudally towards the ipsilateral nipple and slightly medially. End point in a nerve stimulator is a motor response in ulnar distribution side of hand with an output less than or equal to 0.5mA. To avoid intra vascular injection aspiration done every 3-5 ml of the drug injected.within one ml of injection muscular twitch disappear. The solution should flow without resistance. High resistance or pain on injection may indicate intraneural injection and the needle must be repositioned.

Volume of local anaesthetic (either 1% lignocaine or 0.25% Bupivacaine) that can be used is 25-40 ml depending on the weight of the patients. When large volumes are used the sheath may be felt to distend during injection and is easily distinguished from the

subcutaneous swelling of an extra fascial injection. To encourage the spread proximally, digital pressure distal to the needle point may be used and digital pressure proximal to needle insertion point may help to encourage distal spread.

COMPLICATIONS

a) Related to procedure

- Vessel puncture - Haematoma formation
- Pneumothorax
- Neuropraxia

b) Related to Local anaesthetic

- Intravascular injection
- Circumoral numbness
- Convulsions
- Cardiac arrest

5. PHYSIOLOGICAL CONSIDERATIONS[4]

International association for the study of pain has defined pain as "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".

Pain perception requires a noxious stimulus which is transformed from its native form by the activated nociceptors into electrical signals which are then transmitted along the corresponding nociceptive fibres. These fibres in turn synapse onto second order neurons in the spinal cord. These interneurons are located in the dorsal horn. It is at these interneurons where the initial modulation of nociceptive input occurs. From the spinal cord nociceptive input is transmitted to the brain stem, thalamus and cortex.

Peripheral neuroanatomy of nociception

C and A fibres are the main peripheral nociceptors. The skin, joints and periosteum are richly innervated with C and A nociceptors as well as the non nociceptive AB sensory fibres.

A fibres are responsible for the sensation of first pain, the initial sharp pain experienced following an injury. C fibres are unmyelinated and are responsible for second pain, the slowly building throbbing burning pain experienced following an injury.

Classification of sensory fibres

Sensory receptors	Speed of transmission	Sensory function	Myelination
C fibres	0.5 – 2 m/sec1	Noxious chemical, mechanical, thermal activation (slow burning second pain)	Unmyelinated
Aα fibres	70 – 120m/sec	noxious chemical thermal, mechanical stimuli (sharp fast, first pain)	slightly myelinated
Aβ fibres	30 – 70 m/sec	nonpainful, light touch, pressure, vibration, proprioception	heavily myelinated
Aγ fibres	30 – 70 m/sec	proprioception, motor to muscle spindle	myelinated
Aδ fibres	12 – 30 m/sec	pain, cold, touch	myelinated
B fibres	3- 15 m/sec	pre ganglionic autonomic (sympathetic)	myelinated

Peripheral neurochemistry and neurotransmitters

Commonly released inflammatory mediators implicated in pain and hyperalgesia include bradykinins, potassium, substance-P, cytokines, histamine, serotonin and prostaglandins. These peripheral

neurotransmitters either activate or sensitize the peripheral nociceptors to pain.

Peripheral neurochemistry of Algogenic agents:

Algogenic agent	Action on nociceptors
Bradykinin	activates
Substance P	sensitizes
Potassium	activates
Hydrogen	activates
Arachidonic acid	sensitizes
Cytokines	sensitizes
Serotonin	sensitizes
Noradrenaline	high concentration activates and sensitizes after injury.

Physiology of nerve conduction

Neurons are the basic building blocks of the nervous system that responds to various stimuli. Integration and transmission of nerve impulses are specialized functions of neurons.

All peripheral nerves are elongated axons of neurons situated centrally. A typical peripheral nerve consists of bundles of motor, sensory and other fibres enclosed in the outermost covering called epineurium. Each nerve fibre in a bundle is enclosed in a layer of

neurilemma or the axonal membrane. Depending upon the presence or absence of myelin sheath, it can be a myelinated nerve fibre or unmyelinated fibre.

The axonal membrane itself is made up of a bimolecular lipid palisade, interspersed with large protein molecules. The membrane lipids are largely phospholipids composed of a polar head group and a non polar hydrocarbon tail.

The primary function of the cell membrane is to separate the extracellular from the intracellular environment. The major difference between these two environments is the ionic concentration. This disequilibrium provides the means for impulse conduction.

The most important ions in this respect are sodium and potassium. A membrane bound protein sodium potassium ATPase maintains normal resting equilibrium potential between -50mv to -90mv by pumping potassium ions into the cell and sodium ions out of the cell. A positive ion gradient from inside the membrane to outside causes electronegativity inside the membrane.

During nerve conduction the following changes occur in the cell membrane.

In the resting phase:

There is a potential difference across the membrane the inside is negative, due to a higher concentration of sodium ions outside than inside the cell.

Na^+ moves in and K^+ moves out of cells but because of more K^+ channels opened at rest, K^+ permeability is greater than Na+ permeability. Therefore K+ channels maintain the resting membrane potential.

Depolarization phase:

During excitation, Na+ channels in the cell membrane open briefly allowing sodium ions to flow into the cell, thereby depolarizing the membrane.

Repolarization Phase:

During this phase, opening of voltage gated K+ channel occurs, results in passing of potassium ions out of the cell to restore electrical neutrality.

Restoration Phase:

During this phase, sodium ions return to the outside and potassium ions re - enter the cell.

Distribution of ion channels in Myelinated Neurons:

Voltage gated Na+ channels are highly concentrated in the nodes of Ranvier and the initial segment in myelinated neurons.

The initial segment and in sensory neurons, the first node of Ranvier are the sites where impulses are normally generated and the other nodes of Ranvier are the sites to which the impulses jump during saltatory conduction.

The sodium channel is believed to be an integral membrane spanning protein. Depolarization of the cell induces a configurational change on the sodium channel which causes it to open and allow ion passage. In many myelinated neurons, the Na+ channels are flanked by K+ channels that are involved in repolarization.

Action of Local Anaesthetics on Nerve Fibres[5]:

The primary action of local anaesthetics on the nerves is electrical stabilization. The large transient increase in permeability to Na+ ions necessary for propagation of the nerve impulse is

prevented. Thus the resting membrane potential is maintained and depolarization in response to stimulation is inhibited.

Local anaesthetics block sodium conductance by:

1. binding of local anaesthetics to sites on voltage gated Na+ channels prevents opening of the channels by inhibiting the conformational changes that underlie channel activation.

2. local anaesthetics produce nonspecific membrane expansion. There is an unfolding of membrane protein together with a disordering of the lipid component of the cell membrane with consequent obstruction of the sodium channel.

PAIN PATHWAY

SPINAL CORD

The gray matter of the spinal cord is divided into ten lamina with lamina I – IV representing the dorsal horn. The dorsal horn is capped by the Lissauer's tract which consists of branches of cutaneous A and C – fibres and few visceral afferents.

Nociceptive fibres terminate in the superficial layers of lamina I & II while the non-painful myelinated fibres terminate in the deeper layers of lamina III, IV. Lamina II has the highest concentration of opioid receptors in the spinal cord. Modulation and inhibition of

nociception may occur at this level through the use of opioids (systemic and neuraxial).

Ascending sensory pathways

Peripheral sensory neurons synapse onto the secondary interneurons of the dorsal horn. The axons of the non nociceptive secondary neurons travel isobilateral in the dorsal columns of the spinal cord as fasciculus cuneatus (upper body through T6) and fasciculus gracilis (lower body below T6) and synapse in the thalamus.

The axons of the nociceptive secondary neurons after synapsing travel contra laterally in the anterolateral aspects of the spinal cord as the neospinothalamic and paleospinothalamic tract.

Neospinothalamic tract carries fine discrimination of pain e.g. location, intensity, and first pain.

Paleospinothalamic tract responds to noxious stimuli. The paleo spinothalamic tract synapses in the thalamus, hypothalamus and limbic system and plays a role in emotional aspects of pain via limbic system. The thalamus has multiple connections to limbic system and cortex.

Descending inhibitory pathways

The descending controls of pain project specifically onto laminas I, II, V of the dorsal horn from mesencephalon, raphenuclei and reticular tract. The mesencephalon is rich in opioid receptors. This area sends excitatory transmissions to the rostroventral medulla which sends noradrenaline and serotonin inhibitory tracts via the dorsolateral funiculus to lamina I, II, V of spinal cord.

The noradrenaline and serotonin fibres mediate transmission between the primary afferents and the secondary neurons of the dorsal horn. Increased activity of these fibres leads to increased inhibition of pain transmission.

Location of opioid receptors (central)

Opioid receptors are found in the various regions in CNS namely, cerebral cortex, limbic cortex (anterior and posterior amygdale, hippocampus) hypothalamus, medial thalamus, mid brain, periaqueductal gray matter, extrapyramidal areas, substantia gelatinosa and sympathetic preganglionic neurons.

Opioid receptors are also found in the cardiac sympathetic fibres, cardiac branches of vagus, adrenal medulla, and gastro intestinal tract.

6. BASICS OF NERVE LOCATOR[6, 7]

Perivascular technique and elicitation of paraethesia had been the classical methods for locating nerves in peripheral nerve blocks. Peripheral nerve locator technology is a newer one, utilizing objective end points for effective nerve localization.

Peripheral nerve locator is used to elicit Evoked Motor Response (EMR). They are used to assess functioning of Neuromuscular (NM) junction. The other name for Peripheral nerve locator (PNL) is Peripheral nerve stimulator (PNS) .When the high intensity current is used to assess the NM junction function through cutaneous electrodes it is called as PNS. When low intensity current is used to locate the nerve it is called peripheral nerve locator.

Physiological basis of PNL Technology

The ability of a nerve locator to evoke a motor response depend on i) intensity of current

ii) duration of current

iii) polarity of stimulating current used

iv) needle nerve distance.

Assuming a square pulse of the current is used to stimulate the nerve the total charge applied is the product of intensity of current and duration of the pulse.

Rheobase and Chronaxie:

Rheobase is the minimal current required to stimulate the nerve with a long pulse width.

Chronaxie is the duration of the stimulus required to stimulate at twice the rheobase.

$$I = Ir\ (1+c/t)$$

Where I - current required, C - chronaxie,

Ir - rheobase, t - duration of stimulus.

Chronaxie is useful when comparing different nerves or nerve fibre types. The larger fibres are more readily stimulated than the smaller fibres. It is possible to stimulate the larger Aα motor fibre without stimulating the smaller Aδ or C -fibres responsible for pain.

Chronaxies of Peripheral Nerves

* A-α → 50 -100 μ seconds
* A-δ → 170 μ seconds
* C-Fibres → 400 μ seconds

Principles of peripheral nerve stimulation:

i) Preferential cathodal stimulation:

Significantly less current is needed to obtain a response to nerve stimulation when cathode is adjacent to the nerve, rather than the anode.

ii) Variation of stimulus intensity with varying needle nerve distance.

Stimulation intensity will be variable as determined by Coulomb's law.The relationship between the stimulus intensity and the distance from the nerve is governed by Coulomb's law

$I = K (Q/r^2)$

Where I is the current required to stimulate the nerve, K is a constant, Q is the minimal current needed for stimulation, and r is the distance from the stimulus to the nerve.. A very high stimulus current is required to stimulate the nerve when the needle tip is far away from the nerve.

Components of peripheral nerve locators

- Oscillator
- Display
- Constant current generator
- Controls.

Characteristics of an ideal PNL

1) Constant current output:-

The constant current designs of the locator allows for an automatic compensation for changes in tissue or connection impedance during nerve stimulation assuring accurate delivery of the specified current.

2) Options for different pulse width:-

Shorter pulse width corresponds to the chronaxie of motor fibres in a mixed peripheral nerve. Wider pulse width (>100μ sec) is useful for stimulating a sensory nerve or a nerve with compromised conduction i.e. Diabetic neuropathy.

3) A wide range of current output - 0.01 to 5.0 mA:-

A higher current output is needed for patients with neuropathy and sensory nerve stimulation.

4) Digital display of the delivered current.

5) Variable current output dial.

6) Clearly identifiable polarity

7) Disconnect indicator - shows circuit connection status

8) Battery indicator

9) Stimulating frequency:-

If the stimulating frequency is higher, it allows faster manipulation of the needle.

Clinical points to be noted while using PNL:

An effective use of PNL technology mandates knowledge of anatomy with respect to

a) Optimal needle insertion site to achieve needle tip – target nerve contact.

b) Muscle innervations scheme of the targeted nerve to identify desired evoked motor response (EMR).

c) Ability to differentiate desired EMR from the alternate EMR elicited by the stimulation of adjacent muscle and collateral nerves.

d) The relationships of adjacent neuromuscular structures generating those alternate EMR to the targeted nerve.

e) The highest rate of success is attained when a brisk EMR occur between 0.2-0.4mA.

An EMR at currents higher than 0.5mA may result in failed block because the needle tip is too far from the nerve. A brisk EMR at stimulating current lower than 0.1mA may risk nerve damage because of the possibility of an intraneural injection.

Peripheral Nerve Locator Settings:

1) Mixed nerve (most PNB)

Current → 1 mA

Current duration (Pulse with) → 0.1ms

Frequency → 1-2 HZ

2) **Sensory nerve** (e.g. lateral cutaneous and saphenous nerve)

Current → 2-5 mA

Current duration → 1ms

Frequency → 1 Hz

3) **Diabetic neuropathy (PNB)**

Current → 2 mA

Current duration → 0.3 ms

Frequency → 1-2 Hz

APPROPRIATE EVOKED MOTOR RESPONSE FOR EACH PNB

PNB Technique	Optimal EMR
Interscalene	Flexor: Deltoid, Biceps, Pectoralis major Extensor: Triceps, Brachioradialis, Wrist extensors (EMR of > 2 muscles)
Deep Cervical plexus	Rhomboids, Shoulder girdle
Infraclavicular	Muscles of wrist and hand Radial – extension of wrist/ fingers. Median – flexion of wrist / fingers. Ulnar – adduction of thumb / 4^{th} and 5^{th} finger flexion.
Femoral	Quadriceps – patellar tap
Sciatic	Inversion, plantar flexion of the foot

7. PHARMACOLOGY OF BUPIVACAINE[8, 9, 10]

BUPIVACAINE

Bupivacaine is a local anesthetic agent with long duration of action

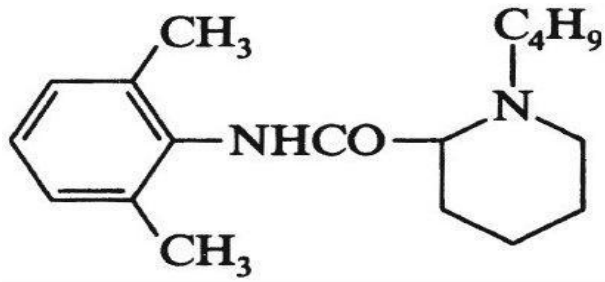
Pharmacology

Bupivacaine hydrochloride is 2 piperidine carboxamide, 1 butyl N-2, 6 dimethyl phenyl, monohydrochloride, and monohydrate.

Bupivacaine molecule is a tertiary amine separated from an aromatic ring system that is a benzene ring by an chain. The tertiary intermediate amine is a base that is a proton acceptor. The chain contains an amide linkage (-NHCO-) hence it is classified as an amino amide compound. This amide linkage contributes to the anesthetic potency.

The aromatic ring system gives a lipophilic character to its portion of molecule whereas the tertiary amine end is relatively hydrophilic.

STRUCTURE



Structure - Activity relationship:

Bupivacaine being more lipophilic (because of butyl group) it is very potent and produces longer lasting blocks.

PHARMACODYNAMICS

Mechanism of action:

The uptake of the drug by the tissues is largely due to lipophilic absorption. This shifts effective pKa downward and thereby favors the neutral base form.

Bupivacaine blocks impulses by reducing the currents through voltage-activated Na+ channels. The inhibition is not specific; however, K+ currents are also reduced. Binding of bupivacaine to sites on voltage gated Na+, channels prevents opening of the channels by inhibiting conformational changes.

It is similar to that of any other local anaesthetics. The primary action of local anaesthetics is on the cell membrane of the axon, on which it produces electrical stabilization. The large transient increase in permeability to sodium ions necessary for propagation of the impulse is prevented. Thus the resting membrane potential is maintained and depolarization in response to stimulation is inhibited. The mechanism by which local anaesthetics block sodium conductance is as follows:

a) Local anaesthetics in the cationic form act on the receptors within the sodium channels, on the cell membrane and blocks it. The local anaesthetic can reach the sodium channel either via the lipophilic pathway directly across the lipid membrane, or via the axoplasmic opening. This mechanism accounts for 90% of the nerve blocking effects of amide local anaesthetics.

b) The second non specific mechanism of action is by membrane expansion.

PHARMACOKINETICS:

- Pka 8.1
- Bound in plasma 95%
- Clearance 7.1 – 2.8 ml/kg/min

- Volume of distribution 0.4 – 0.9 litres / kg
- Half life 1.2 – 2.4 hours
- Peak time 0.17 – 0.5 hours
- Peak concentration 0.8 micrograms/ml
- Toxic plasma concentration >1.5 micrograms/ml
- Clearance – 0.47 litres /min
- Metabolism – Liver by dealkylation to Pipecolyloxilidine
- Excretion – 5% by the kidney as unchanged drug and the rest as metabolites.

TABLE: 4. Dosage and concentration of Bupivacaine in various block

Type of Block	Concentration	Dosage ml	Dosage mg
Local infiltration	0.25 – 0.5%	5 – 20 ml	upto 175mg
Brachial plexus block	0.25 – 0.5%	20 – 40 ml	75 – 225mg
Intercostal nerve block	0.25 – 0.5%	3 – 5 ml	15 – 20 mg per each nerve
Epidural block	0.25 – 0.5%	15 – 20 ml	50 – 200mg
Caudal block	0.25 – 0.5%	15 – 30 ml	75 – 150mg
Subarachnoid block	0.5%	2 – 4 ml	10 – 20mg

DRUG DOSAGE[11, 12]

Bupivacaine upto 3mg/kg.

Addition of epinephrine to bupivacaine has no effect on its duration of action but it delays absorption of local anaesthetic due to vasoconstriction from the site of administration.

Toxicity of Bupivacaine

It is relatively free of side effects if administered in an appropriate dosage. It is more cardiotoxic than Lignocaine and this is made worse by hypoxia, hypercapnia, acidosis and pregnancy.

1. Central nervous system toxicity

CNS is more susceptible to bupivacaine. The initial symptoms involve feeling of light headedness and dizziness followed by visual and auditory disturbance. Disorientation and occasional feeling of drowsiness may occur. Objective signs are usually excitatory in nature which includes shivering, muscular twitching and tremors; initially involving muscles of the face (perioral numbness) and part of extremities. At still higher doses cardiovascular or respiratory arrest may occur. Acidosis increases the risk of CNS toxicity from

Bupivacaine, since the elevation of $PaCO_2$ enhances cerebral blood flow, so that more anesthetic is delivered rapidly to the brain.

2. Cardiovascular system toxicity

Bupivacaine depresses rapid phases of depolarization (Vmax) in purkinge fibres and ventricular musculature to a greater extent than lignocaine. It also decreases the rate of recovery from a dependent block than that of lignocaine. This leads to incomplete restoration of Vmax between action potential at high rates, in contrast to complete recovery by lignocaine. This explains why lignocaine has antiarrhythmic property while bupivacaine has arrhythmogenic potential. High level of bupivacaine prolongs conduction time through various parts of heart and extremely high concentration will depress spontaneous pacemaker activity, resulting in bradycardia and arrest. Cardiac resuscitation is more difficult following bupivacaine induced cardiovascular collapse and hypoxia along with acidosis which markedly potentiates cardiac toxicity. Bretylium raise the ventricular tachycardia threshold that was lowered by bupivacaine but not by lignocaine.

The cardiovascular collapse / central nervous system ratio for bupivacaine is 3.7 ± 0.5.

3. Respiratory system:

Respiratory depression may be caused if excessive plasma level is reached which in turn results in depression of medullary respiratory center.

4. Autonomic nervous system:

Myelinated preganglionic beta fibers have a faster conduction time and are more sensitive to the action of local anesthetic including bupivacaine. Involvement of preganglionic sympathetic fibers is the cause of widespread vasodilatation and consequent hypotension that occurs in epidural and paravertebral block. When used for conduction blockade all local anesthetic agents particularly bupivacaine produces higher incidence of sensory blockade than motor fibers.

Treatment of adverse reaction:

Treatment is mainly symptomatic i.e. maintaining circulation with IV fluids and vasopressors if required to restore the cardiovascular stability and to support ventilation with oxygen or

controlled ventilation. Convulsions may be controlled with diazepam (0.1- 0.2 mg/kg) or thiopentone (2-3 mg/kg) or a muscle relaxant and controlled ventilation with oxygen. Corticosteroids, if allergic reactions are suspected. Treatment of ventricular fibrillation and tachycardia by amiodarone (5mg/kg IV), bretylium (5mg/kg) or defibrillation (2-6 joule/kg).

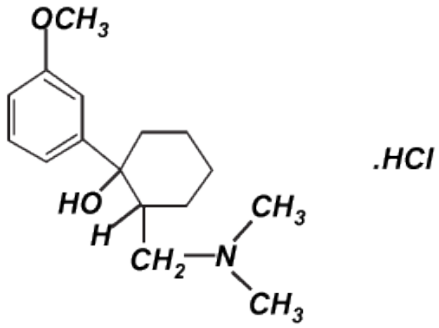
Role of additives:

i. Adrenaline: Onset time reduced and duration prolonged.
ii. Sodium bicarbonate: Onset time reduced and duration variable.
iii. Clonidine: Onset time reduced and duration prolonged.
iv. Hyaluronidase: Onset time reduced and duration variable.
v. Opioids: Onset time reduced and duration prolonged. Reports controversial.
vi. Midazolam: Onset time reduced and duration prolonged.

8. PHARMACOLOGY OF TRAMADOL[13, 14, 15, 16]

Tramadol is an analgesic with unique dual mechanism. It is a synthetic 4 – phenyl piperidine codeine analogue. Tramadol is a racemic mixture of 2 enantiomers.

Structure of Tramadol



• Chemical name: Tramadol belongs to the aminocyclohexanol group.

Mechanism of action

Tramadol has both opioid and non-opioid actions

1. Tramadol has a low affinity for opioid receptors. It acts as a selective μ-receptor agonist, but also binds weakly to kappa and delta receptors.

2. Non-opioid mechanism is by monoaminergic pathway. It inhibits noradrenaline and 5-hydroxy tryptamine (serotonin) neuronal reuptake and facilitates serotonin release. The two enantiomers of

tramadol i.e., tramadol (+) and tramadol (-) have complementary and synergistic anti-nociceptive interaction. Tramadol (+) has greater affinity for μ receptors and inhibits serotonin reuptake. Tramadol (-) inhibits norepinephrine reuptake. The synergistic effect of both these enantiomers may be responsible for its low potential for the development of tolerance, dependence, and abuse and production of analgesia with the absence of ventilatory depression. Interestingly, the racemate may produce less sedation and gut inhibition than either enantiomer alone.

Pharmacodynamics

Effects on respiration

In clinically recommended doses, tramadol is unlikely to produce relevant respiratory depression.

Effects on cardiovascular system

Tramadol increases transiently heart rate, both systolic and diastolic blood pressure; it increases peripheral vascular resistance, decreases pulmonary arterial resistance, and exerts a negative inotropic effect on left ventricular myocardium.

Tolerance and dependence

Tolerance is minimal. It has low physical and psychological dependence.

Other effects

Tramadol in clinical dosage has no effect on plasma histamine levels and it does not cause any systemic anaphylactoid reactions.

Pharmacokinetics

- Bioavailability

  Oral 68 %(70 – 75%)

  Intramuscular 100%

- Elimination T1/2 6hours (4.5 – 7.5%)
- Metabolite 7.5hours
- Percentage of drug bound in plasma 20%
- Clearance 8ml/min/kg (6 – 12ml)
- Volume of distribution 2.7litres/kg (2.3 – 3.9)
- Onset of analgesia 1 hour
- Peak time 2.3 ± 1.4 hours
- Peak concentration 592 ± 178 nanogram /ml in blood

- Metabolism – Liver by O- demethylation to O-desmethyl tramadol which shows 200 times higher affinity for μ receptors.
- Elimination – 15 – 30% as unchanged drug by kidney.

Therapeutic efficacy

On intravenous administration, tramadol is equivalent to pethidine, l/5th as potent as nalbuphine, 1/10th as potent as morphine.

Dosage

Tramadol can be given in doses of 50-100 mg upto 4 times a day. Total daily dose should not exceed 400mg for adults. In children > l year of age the dosage is 1 – 2 mg/kg.

Routes of administration

Oral, parenteral, epidural, rectal, caudal.

Adverse effects: Mild, transient and rare

1. CNS: Nonspecific CNS irritation, dizziness, sedation, euphoria, dysphoria.
2. GIT: Nausea, vomiting, constipation, GI irritation
3. ANS: Dry mouth, sweating (due to its monoaminergic effects)

4. CVS: Orthostatic hypotension, tachycardia

5. Others: Motor weakness, urinary retention

Respiratory depression with tramadol is less than that with morphine. Respiratory depression is unusual in recommended doses and was not found in neonates whose mother had been given tramadol. The advantage of tramadol over other opioids with respect to less respiratory depression is limited by lack of efficacy of tramadol in severe pain.

Drug interactions

Tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRI), neuroleptics: tramadol when given to patients on these drugs decreases the seizure threshold. Concomitant administration of tramadol and SSRI causes serotonin syndrome.

Concomitant administration of tramadol with monoamino oxidase inhibitors causes hypertensive reactions. Quinidine inhibits tramadol metabolism. Hence serum tramadol concentration increases, when these 2 drugs are used. Carbamazepine enhances tramadol metabolism, hence tramadol halflife is decreased as much as 50%. When these two drugs are used concomitantly, tramadol dose should be increased.

Overdosage:

Symptoms are similar to other opioids. Miosis, vomiting, coma,

respiratory depression, respiratory arrest and cardiovascular collapse. Opioid antagonist naloxone will reverse coma and respiratory depression.

Advantages:

1. Can be given through different routes - oral, parenteral etc.

2. Less respiratory depression

3. Less dependence, abuse, tolerance

4. Less secretion in the milk of lactating mother.

5. Freely available. No narcotic prescription restriction.

6. Comparatively cheap.

Indications:

1. Tramadol is indicated for moderate to severe pain in adults. Tramadol 50-150 mg IV was equivalent to morphine 5-15 mg but a preservative free preparation had 1/13 of the potency of morphine extradurally.

Despite being relatively less potent than pure opioids, tramadol has achieved efficacy when used to treat moderate pain after surgery. Because of its efficacy as an analgesic, tramadol is considered to be effective in step two of the world health organization guideline for treatment of patients with cancer pain.

2. In peripheral nerve blocks along with local anaesthetics to prolong the duration of anaesthesia as well as analgesia.

Contraindications:

As tramadol enhances monoaminergic transmission, the drug is contraindicated in patient receiving mono-amino oxidase inhibitors and caution advised in patients with epilepsy. Not recommended in children <1 year.

9. REVIEW OF LITERATURE

1. **Suman Chattopadhyay et al 2007**[17] conducted a prospective double blind study in supraclavicular brachial plexus block to evaluate the effect of weak opioid tramadol with nonopioid mechanisms of action, improves post operative analgesia when used as an additive along with bupivacaine in 70 patients who underwent surgery of various upper limb surgeries. These patients are randomly allocated into 2 equal groups so that 35 patients received inj. bupivacaine (0.25%) - 38 ml + 2 ml normal saline (Group C) and the remaining 35 received inj. bupivacaine (0.25%) - 38 ml +tramadol 100 mg (2 ml) (Group T) [total volume in both group 40 ml].The onset, quality and duration of the block and duration of analgesia was assessed as well as the possible side effects and also the incidence of various complications following the procedure. Earlier the onset of motor (6.1 ± 1.2 vs 8.6 ± 1.4 mins) and sensory blockade(11.2 ± 2.1 vs 18.4 ± 2.5mins) and the duration of pain relief (410.1 ± 95.1 min vs 194.8 + 60.4 min) produced with addition of tramadol was longer and superior in comparison to control group ($p<0.05$). Tramadol group also demonstrated no changes in pulse

rate, blood pressure and spo_2. No side effect was noted in any of the patients. Tramadol is a useful adjuvant for brachial plexus block.

2. **Renu Wakhlo et al 2009**[18] conducted a study on 60 patients to compare the adjuncts- tramadol and butorphanol to lignocaine with adrenaline for onset and duration of block and post operative analgesia for upper limb surgeries following supraclavicular brachial plexus block. All patients received total volume of 30 cc of anaesthetic. Patients were randomly divided into three equal groups so that 20 patients received only lignocaine with adrenaline (1:200,000) 20 cc and 10 cc saline (Group I), next 20 patients received lignocaine with adrenaline + tramadol 100 mg (2 cc) +8cc saline (Group II) and remaining 20 received lignocaine + adrenaline + butorphanol 1 mg. (1 cc) + saline 9 cc (Group III). The onset of sensory and motor block, duration of block and post operative analgesia was compared. Statistical analysis was done by ANOVA test and intergroup comparison done by Bonferroni's t test. It was found that Group II patients had earlier onset and prolonged duration of sensory and motor block while Group III patients had prolonged duration of postoperative analgesia lasting upto mean of 12 hours. Thus tramadol was found to be good agent for hastening the onset

and prolonging the sensory and motor block while butorphanol is suitable agent for prolonged postoperative analgesia.

3. **S.Antonucci et al 2001**[19] compared the adjuvant effects of Clonidine (1.5 mcg/kg), Sufentanil (20 mcg) and Tramadol (100mg) with 0.75% Ropivacaine in brachial plexus axillary blockade among 80 patients of each group20 with control group as normal saline and concluded that the use of tramadol 100mg as adjuvant provides a significant reduction of onset time and provides a prolonged anaesthesia and analgesia with a quality of block similar to that of Clonidine and Sufentanil and incidence of side effects like sedation, bradycardia and hypotension and itch are lower. They concluded that tramadol 100mg may be a useful alternative as adjuvant in periphery block with same effects of other drugs commonly used and a lower incidence of side effects.

4.Sukran Geze et al 2012[20] compared tramadol 100mg and fentanyl 50mcg by adding to 40ml of 0.25% levobupivacaine plus 40mg lignocaine mixture for axillary plexus block by randomized double blind study and concluded that tramadol hydrochloride or fentanyl when added to local anaesthetic mixtures as an adjuvant agent provide better postoperative analgesia in axillary block for

orthopaedic upper limb surgeries. Furthermore tramadol improves the block quality more than fentanyl.

5. **Shrestha BR et al 2007**[21] evaluated the postoperative analgesia of tramadol 2mg/kg and dexamethasone 8mg as admixture to bupivacaine by conducting a prospective, randomized, double blind study in 60 patients. Patients were randomly allocated in to two groups of 30 each. The duration of postoperative analgesia was recorded in both groups using pain VAS score which was determined by maximum VAS score of 8-10 and when patient demands for additional analgesics. Dexamethasone significantly prolonged the postoperative analgesic duration (mean 1028 minutes) which was significant than tramadol (453.17 minutes) ($P < 0.05$).This helps to minimize the cost and provides patient comfort.

6. **Sebastien Robaux et al 2004**[22] designed a prospective, randomized, controlled and double-blind clinical trial to assess and evaluate the time of onset and quality of postoperative analgesia, and occurrence of adverse effects of tramadol in one-hundred patients scheduled for carpal tunnel release surgery. All patients received 1.5% mepivacaine 40ml.The patients were randomly divided into four groups so that 17 patients received isotonic sodium chloride

(Group P), 22 patients received tramadol 40 mg (Group T40), other 20 patients received tramadol 100 mg (Group T100) and remaining 20 received tramadol 200 mg (Group T200).Onset and duration of sensory and motor blocks were not different among groups. The number of patients requesting analgesia in the postoperative period was significantly less in the 3 tramadol groups compared with the placebo group (P=0.02); this was also noted with the placebo and T40 groups compared with the T200 group. No statistical significance was demonstrated between the placebo and the T40 group or the T100 group and the T200 group. Furthermore, there was a significant trend effect among groups applying the Cochran-Armitage tendency test (P=0.003), suggesting a dose-dependent decrease for additional postoperative analgesia requirements when tramadol was added. Side effects did not differ among groups, although they were more frequently recorded in the T groups. Study suggests that tramadol added to 1.5% mepivacaine for brachial plexus block enhances in a dose-dependent manner the duration of analgesia with acceptable side effects. However, the safety of tramadol has to be investigated before allowing its use in clinical practice.

7. **Olfa Kaabachi et al 2009**[23] conducted a prospective randomized study to evaluate the effect of varying doses of tramadol as an adjuvant to axillary block with lidocaine 1.5% (epinephrine 1/2, 00,000). Three groups were allotted randomly, control group received 4ml saline, TL group received100mg tramadol and 2ml saline and TH group received 200mg tramadol along with lidocaine. The results showed that the addition of 200mg tramadol prolonged the analgesic duration with a delayed onset time.

8. **Stephan Kapral et al 1999**[24] studied in patients randomly assigned to receive either 40ml of 1% mepivacaine with 2ml of isotonic sodium chloride solution (Group A), 40 ml of 1% mepivacaine with 100mg tramadol or 40ml of 1% mepivacaine with 2ml of isotonic sodium chloride and 100mg tramadol intramuscularly and evaluated that when tramadol 100mg added to 1% mepivacaine prolongs the duration of blockade without any adverse effects in brachial plexus axillary block. Tramadol may be a useful alternative to epinephrine and clonidine as an adjuvant to local anaesthesia for axillary block.

9. **F. Alemanno et al 2012**[25] studied in 120 patients allocated in 3 groups. Group P received 0.4ml/kg of 0.5% levobupivacaine plus isotonic sodium chloride and isotonic sodium chloride intramuscularly. Group T_{PN} (perineural tramadol) received 0.4ml/kg of 0.5% levobupivacaine plus 1.5mg/kg tramadol perineurally and isotonic sodium chloride intramuscularly and last Group T_{IM} received 0.4ml/kg of 0.5% levobupivacaine plus isotonic sodium chloride perineurally and 1.5mg/kg tramadol intramuscularly and concluded that the T_{PN} Group Tramadol and 0.5% levobupivacaine after single - shot interscalene block in patients undergoing shoulder arthropalsty extends the duration of postoperative analgesia without significant side effects.

10. **Ravi Madhusudhana et al 2011**[26] studied and concluded that there are significant beneficial effects on duration of sensory, motor blockade and VAS scores when tramadol 50mg and fentanyl 50mcg added to local anaesthetics 0.75% ropivacaine in supraclavicular brachial plexus block.

11. **W. Kunapis et al 2010**[27] studied and concluded that adding tramadol 2mg/kg to bupivacaine 1.3mg/kg in brachial plexus block for orthopedic procedures in dogs provides faster onset and highly

effective quality and prolonged duration of analgesia with superior pain relief and less requirement of isoflurane to maintain anaesthesia.

Tramadol in other regional anaesthetic procedures:

12. **Ahsan K. Siddiqui et al 2008**[28] studied and confirmed that tramadol 100mg is an effective dose that shortens the sensory block onset, improves tourniquet tolerance and also reduces the intraoperative analgesia consumption when added as adjuvant to intravenous regional anaesthesia with lignocaine.

13. Ahed Zeidan et al 2008[29] studied and concluded that intraarticular admixture of 100mg Tramadol hydrochloride with 0.25% Bupivacaine provided longer duration of analgesia postoperatively with significant lower requirement of rescue analgesia and not associated with any adverse effects when compared to bupivacaine group and tramadol group. This was also associated with earlier recovery of unassisted ambulation and home discharge.

14. **Shrestha BR et al 2005**[30] studied and concluded that when tramadol 2mg/kg used as an admixture with Local Anaesthetics bupivacaine(1mg/kg) and lignocaine (<2mg/kg) can produce an

average duration of postoperative analgesia of 18 hours following circumcision in the children.

15. **Yu Chuan Tsai et al 2001**[31] studied direct tramadol application on sciatic nerve inhibits spinal somatosensory evoke potentials in rats and concluded that direct application of tramadol on the sciatic nerve inhibits SSEP in a dose dependent and reversible manner that is not affected by naloxone and their data suggest that tramadol exerts a local anaesthetic type effect on peripheral nerves.

16. **E Kargi et al 2008**[32] studied tramadol as a local anaesthetic in tendon repair surgery of the hand and concluded that 5% tramadol plus adrenaline had local anaesthetic effects similar to those of lidocaine plus adrenaline when used as infiltration anaesthesia during the surgical repair of injured tendons of the hand. Furthermore treatment with tramadol plus adrenaline was not associated with any local side effects and there was no requirement for additional analgesia during the first 24hour post operation.

17. **Shrestha SK et al 2010**[33] studied and concluded that caudal administration of 1mg/kg tramadol along with 0.5ml/kg of 0.25% bupivacaine improves the quality and prolongs the duration of

postoperative analgesia in children undergoing infra-umbilical surgeries, without any adverse effects.

18. **J Balavenkatasubramnian et al 2008**[34] in his study continuous peripheral nerve block: the future of Regional Anaesthesia? said peripheral opioids – opioid agents are known to exert their effects peripherally. Adding small doses of opioids to local anaesthetic solution for peripheral block have resulted in improvement in the onset time, quality and duration of nerve block. Tramadol has a local anaesthetic effect on peripheral nerves and could provide potentially a synergestic effect in continuous infusion as an additive to local anaesthetic agent.

10. MATERIALS AND METHODS

This study was conducted at Thanjavur Medical College and Hospital, Thanjavur in orthopaedic and plastic surgery theatres.

A prospective double – blinded randomized control study conducted on 60 patients of ASA grade I or II of either sex and age more than 19 years undergoing upper limb surgery under supraclavicular brachial plexus block performed by subclavian perivascular approach with nerve stimulator were included.

The study was started after receiving institutional ethical committee approval and informed written consent from the patients and they were randomly divided into two groups namely-

Group B: 30 patients received 38ml of 0.25 % bupivacaine + 2ml normal saline

Group BT: 30 patients received 38ml of 0.25% bupivacaine + 2ml tramadol (2mg/kg).

Care was taken so that the toxic doses of the local anesthetics were not exceeded according to the weight of the patients.

Inclusion criteria

The following criteria were taken for including the patients in this study.

- ASA Status I and II
- Age between 19 and 72 years
- Weight ≥ 50 kg
- Patients undergoing surgeries in distal end of arm, forearm and hand

Exclusion criteria

- Patient refusal
- Local infections at the site of puncture for block / Sepsis
- Known allergy for the drugs to be studied.
- Coagulation abnormalities
- H/o significant systemic disorders
- Alcohol/drug abuse
- Pregnancy/lactating women
- Chronic analgesic therapy (other than NSAIDS)
- Peripheral neuropathy
- Very obese
- Not fulfilling inclusion criteria

Investigations Required

- Hb%, TC, DC, BT, CT
- Urine routine
- Random Blood sugar
- Serum Urea and Serum Creatinine
- Chest X-ray, ECG
- HIV

MATERIALS

1. Sterile tray for regional blocks
2. Drugs for the block

    0.5% bupivacaine
    Inj. tramadol

    Normal saline

    Distilled water

    2% lignocaine
3. Nerve stimulator with insulated needle
4. Equipments and drugs for resuscitation and conversion to general anaesthesia in the case of block failure.

Methods

Pre Operative preparation

Patients were pre-operatively assessed and the procedure was explained to the patient. Written informed consent was obtained. They were assessed with particular attention to any contraindications.

Postoperative assessment of pain was done using Visual Analogue Scale (VAS) .Patient was explained pre operatively about the visual analogue scale as 0 – No pain and 10 the worst possible pain and was asked the score in visual analogue scale.

Pre medication

Tab. Ranitidine 150mg 2 hours before surgery with sips of water.

Conduct of anaesthesia

On arrival of the patient in the operating room, monitors like pulse oximeter, non invasive blood pressure and ECG were connected and baseline values were recorded. An 18 G intravenous access was obtained in the opposite arm.

40ml prepared solution for Brachial plexus block Supraclavicular approach by the subclavian perivascular technique with nerve locator done.

After evaluation of blocks patients were sedated with Inj. midazolam 0.05mg/kg slow iv along with inj. metaclopramide 10mg iv. Patients were given supplemental O_2 through face mask and intravenous fluids throughout the procedure and they were properly screened from the surgical field.

Parameters observed

1. **Onset of sensory blockade: -** evaluation has done every minute after the performance of the block by Hollman's scale.

   1. normal sensation of pin prick

   2. pinprick felt as sharp but weaker compared with the same area in the other limb.

   3. pinprick recognized as touch with blunt object.

   4. no perception of pin prick.

   Onset of blockade was taken as abolishment of pin prick pain (Hollman's ≥ 3) over the distribution of ulnar and median nerve.

2. **Onset of motor blockade** : Onset of motor blockade was assessed every minute after the block using Bromage three point score

0 – normal motor function with full flexion and extension of elbow, wrist and fingers.

1- decreased motor strength with ability to move fingers and /or wrist only

2- complete motor blockade with inability to move fingers

Attaining a score of 2 was considered as the onset of motor block.

3. **Duration of surgery**: Time taken by the surgeon to do the surgical procedure.

4. **Duration of motor blockade**: When (2) in the three point score changes to (1) the motor blockade is said to reverse. The duration of motor block is noted from the time from score (2) to scale (0).

The pain was assessed using visual analogue scale having 10cm length from 0 to 10.0 – no pain; 10-worst pain. The patient were observed every 30 minutes after the surgery is over till the motor block reverses and there after hourly for 6 hours, 2 hourly for next 6 hours and then 24 hours.

5. **Duration of Sensory block**: Taken from the time of onset of block to first complaint of pain sensation (vas score 1)

6. Duration of analgesia: Taken from the time of the onset of block to appearance of pain requiring analgesia (vas score more than 4).

7. Rescue Analgesia: Time at which VAS score is greater than 4 is noted and patient was given rescue analgesic in the form of inj. Diclofenac sodium intragluteally in the dose of 1.5mg/kg along with inj. ranitidine 50mg given intravenously. Numbers of rescue analgesics in 24hours of postoperative period were recorded.

8. **Vital parameters:**

Pulse rate, blood pressure, saturation were monitored every 5min for first 30min and thereafter 15min till the end of surgery.

9. Perioperative complications and side effects were observed for 24hours

Respiratory Depression

Pneumothorax

Neurological sequale

Nausea

Vomiting

Hypotension

Bradycardia

Sedation

Shivering

Dry mouth

Arrhythmia

Local anaesthetic toxicity

Pneumothorax.

10. Block failure: Failure of the block to be established even after 30 minutes was taken as block failure.Patients in which the block was unsuccessful due to total failure or missed dermatomes which needed general anesthesia or intravenous supplementation were excluded from the study.

11. All the data were subjected to statistical analysis.

11. OBSERVATIONS AND RESULTS

The information collected in our study Group B and Group BT were recorded in a Master Chart. Data analysis was done with the help of computer using SPSS. For statistical analysis students t test was used for comparison between the groups. Using this range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. A 'p' value less than 0.05 was considered statistically significant.

Demographic profile of the patients:

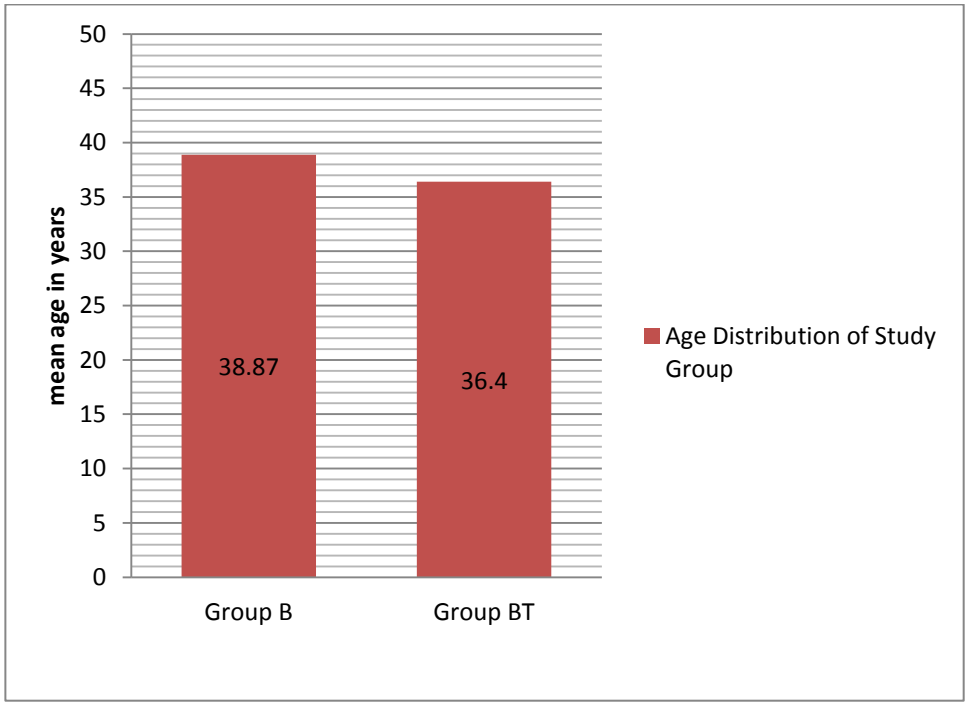
Table 1:

Sl. No.	**Demographic profile**	**Control group (B)**	**Tramadol Group (BT)**
1	No. of patients	30	30
2	Average age (years)	38.87 $\pm$ 13.544	36.40 $\pm$ 11.440
3	Weight (in Kgs)	66.70 $\pm$ 5.914	66.90 $\pm$ 5.026
4	Gender ratio (Male : Female)	17:13	24:6

The above table shows that the average age was 38.87 ± 13.544 years in group B and 36.40 $\pm$ 11.440 years in group BT. Youngest patient in our study group was 19 yrs and oldest was 72 years. The average weights of the patients were 66.70 $\pm$ 5.914 kgs in

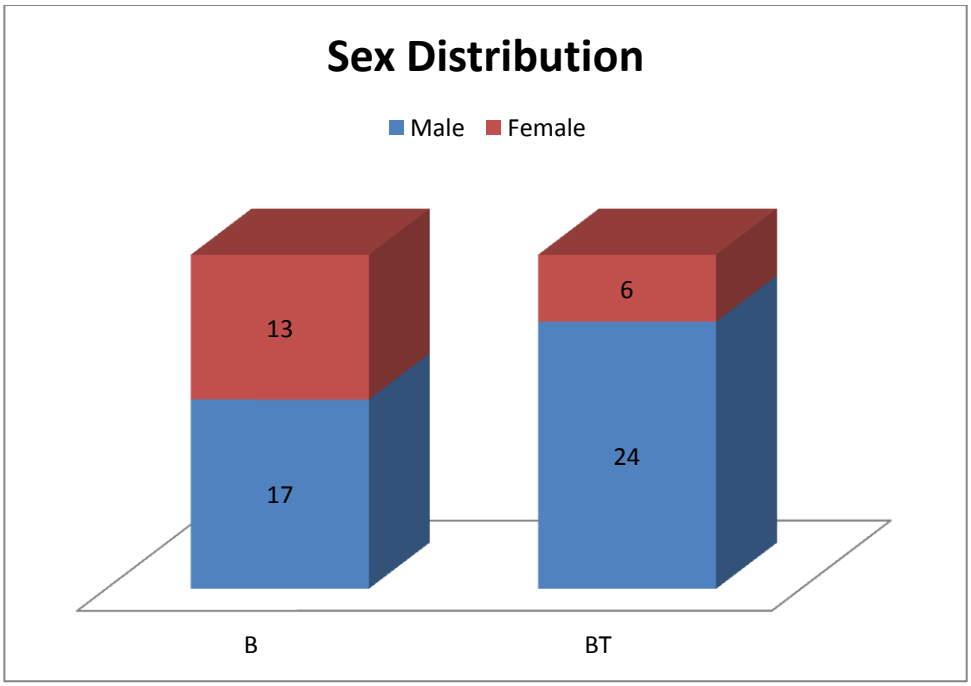
GRAPH 1:

MEAN AGE OF THE PATIENT



GRAPH 2:

SEX DISTRIBUTION OF PATIENTS



group B and 66.90 ± 5.026 kgs in group BT respectively. Majority of the patients in both groups were males. There was no significant difference in age, weight and sex distribution.

Table: 2 Mean Age of the patients (years)

Sl.no	Age	Group		Statistical inference
		B (n=30)	BT (n=30)	
1	Below 30yrs	11 (36.7%)	11 (36.7%)	X^2=3.651 Df=3 .302>0.05 Not Significant
2	31 to 40yrs	8 (26.7%)	11 (36.7%)	
3	41 to 50yrs	7 (23.3%)	2 (6.7%)	
4	51yrs & above	4 (13.3%)	6 (20%)	

Table : 3 Sex distribution

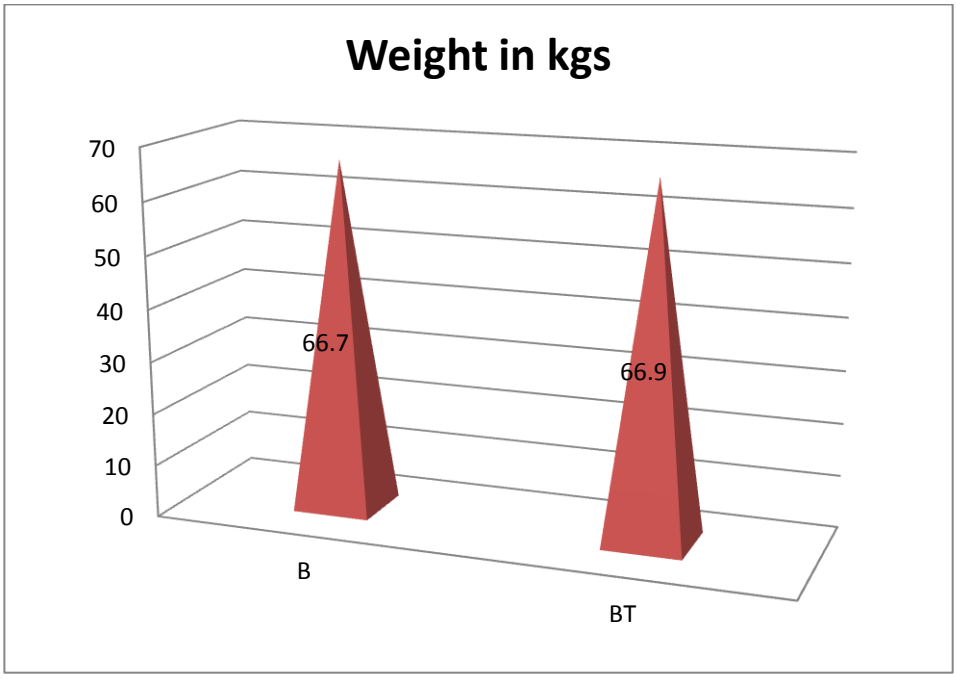
Sex	Group R		Group RC	
	No	%	No	%
Male	17	56.7	24	80
Female	13	43.3	6	20
Total	30	100	30	100
'p'	0.0959>0.05 Not significant			

Table : 4 Mean Weight

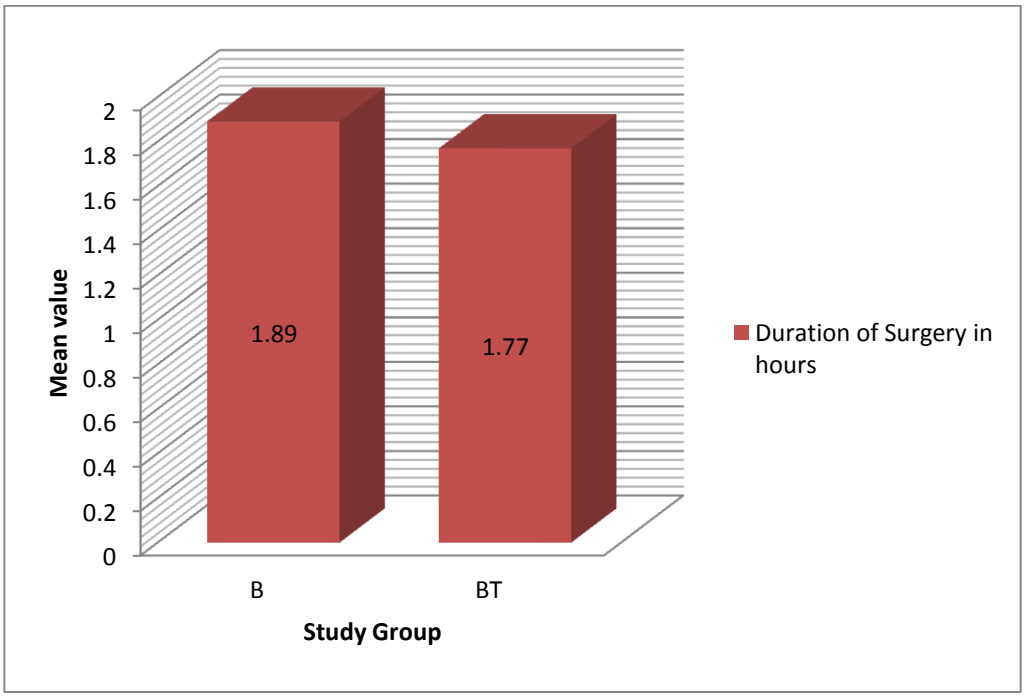
Sl. no	Weight	Mean	S.D	Statistical inference
1	B (n=30)	66.70	5.914	T= -.141 .888>0.05 Not Significant
2	BT (n=30)	66.90	5.026	

GRAPH 3:

MEAN WEIGHT OF THE PATIENTS

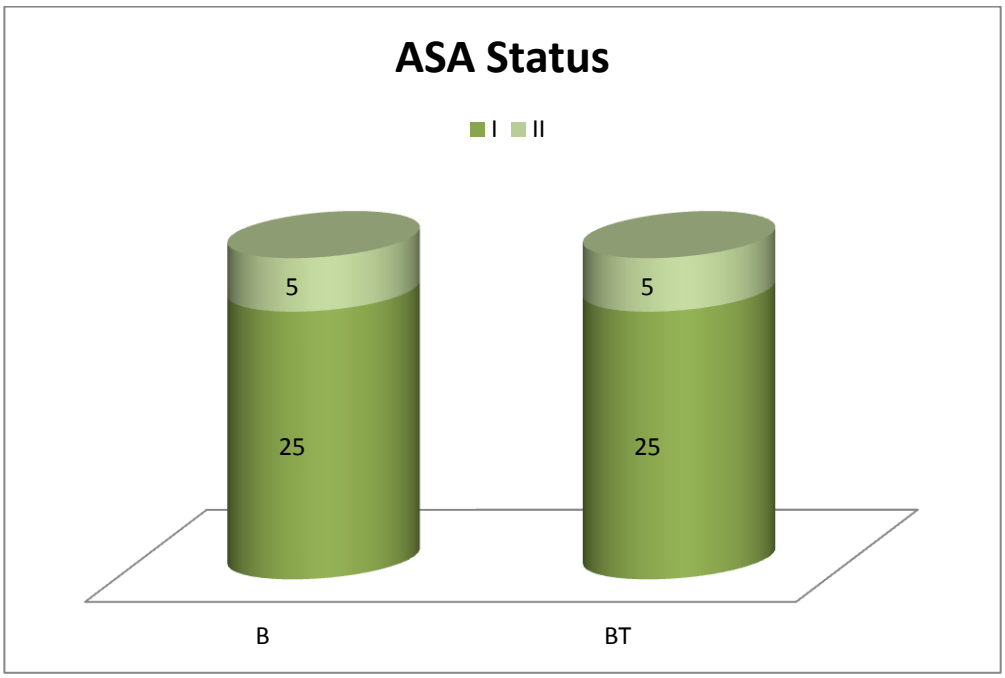


GRAPH 4:
DURATION OF SURGERY



GRAPH 5:

ASA STATUS



Surgical profile of the patients:

Table : 5

	Surgical Profile		
1	ASA Status(I : II)	25:5	25:5
2	Duration of surgery (in hours)	1.89 ± 0.484	1.77 ± 0.388
3	Type of operations (orthopaedic : Plastic surgeries)	(16:14)	(19:11)

Table 6: ASA Status

ASA Status	Group R		Group R C	
	No	%	No	%
I	25	83.3	25	83.3
II	5	16.7	5	16.7
Total	30	100	30	100
'p'	2 >0.05 not significant			

Table 7: Duration of surgery in hours

Duration of surgery in hours	Mean	S.D	Statistical inference
B (n=30)	1.89	.484	T=1.089 .281>0.05 Not Significant
BT (n=30)	1.77	.388	

ASA Status, type and duration of surgery were similar in both groups. The mean duration of surgery was 1.89 ± 0.484 hours in group B compared to 1.77 ± 0.388 hours in BT group. There was no clinical or statistical significance.

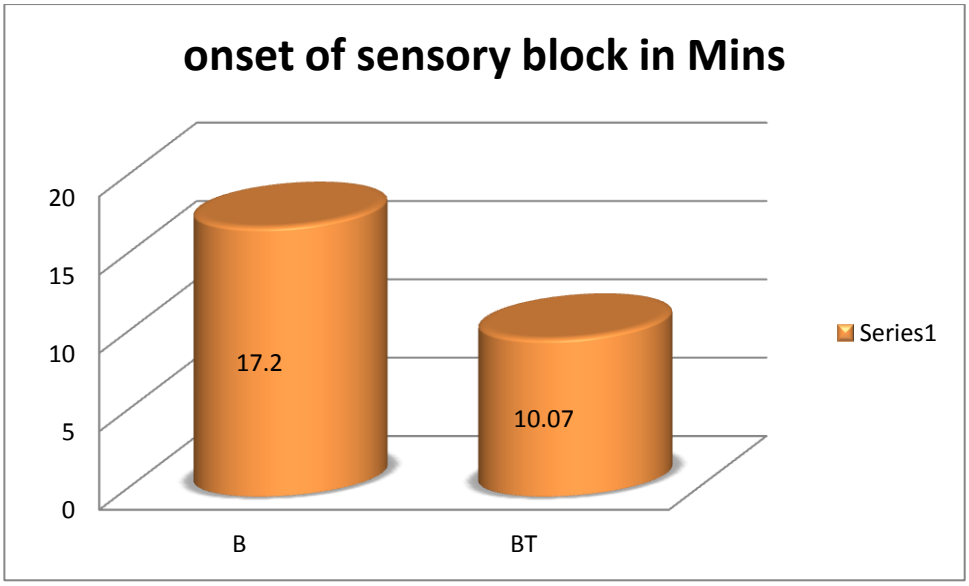
2. **Onset of sensory block between study groups:**

The mean time for onset of sensory block in Group B was 17.20 ± 2.140 and in Group BT was10.07 ± 1.837 .the statistical analysis by students 't' test showed that the time for onset of sensory block in group BT was significantly faster when compared to Group B ($p < 0.05$) as shown in Table 8 and Graph 6.

Table: 8

onset of Sensory block in Minutes	Mean	S.D	Statistical inference
B (n=30)	17.20	2.140	T=13.854 .000<0.05 Significant
BT (n=30)	10.07	1.837	

Graph 6:



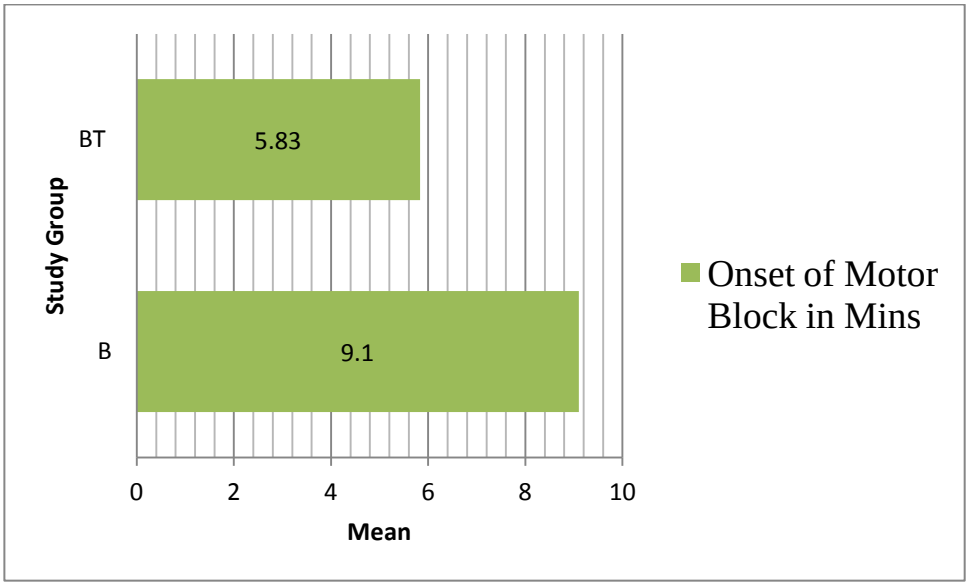
3. **Onset of motor block between study groups:**

The mean time for onset of motor block in Group B was 9.10 ± 1.373minutes and in Group BT was5.83 ± 1.053minutes as shown in table 9 and Graph 7.The statistical analysis by students 't' test showed that the time for onset of motor block in group BT was significantly faster when compared to Group B (p <0.05)

Table: 9

Onset of motor block in minutes	**Mean**	**S.D**	**Statistical inference**
B (n=30)	9.10	1.373	T=10.338 .000<0.05 Significant
BT (n=30)	5.83	1.053	

Graph: 7



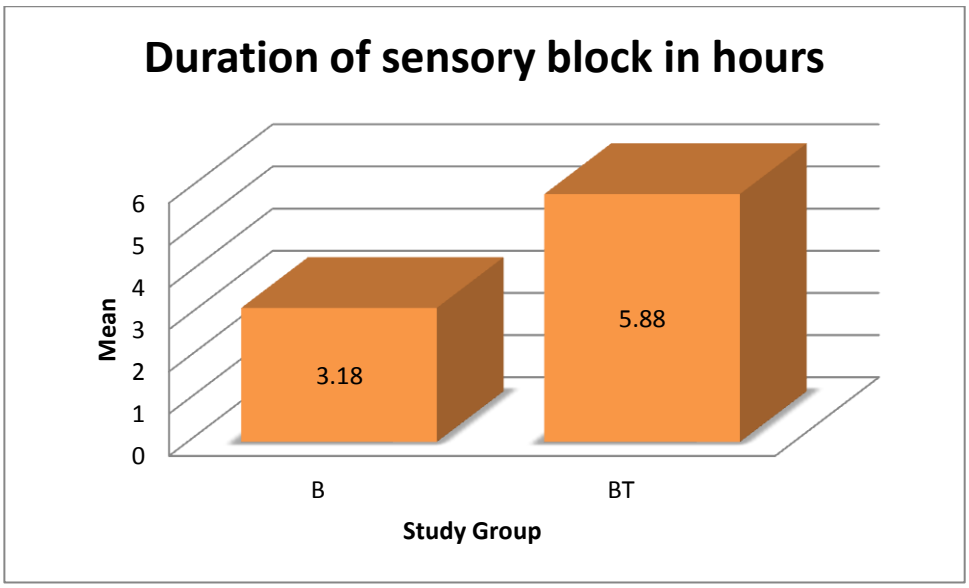
4. **Duration of sensory block in hours:**

The duration of sensory blockade in Group B was 3.18 ± 0.524 hours and in Group BT, was 5.88 ± 0.669 hours as shown table 10, Graph 9.The statistical analysis by students 't' test showed that the time for duration of sensory block in group BT was significantly longer when compared to Group B (.000<0.05)

Table: 10

Duration of sensory block in hours.	Mean	S.D	Statistical inference
B (n=30)	3.18	.524	T=-17.392 .000<0.05 Significant
BT (n=30)	5.88	.669	

Graph:9



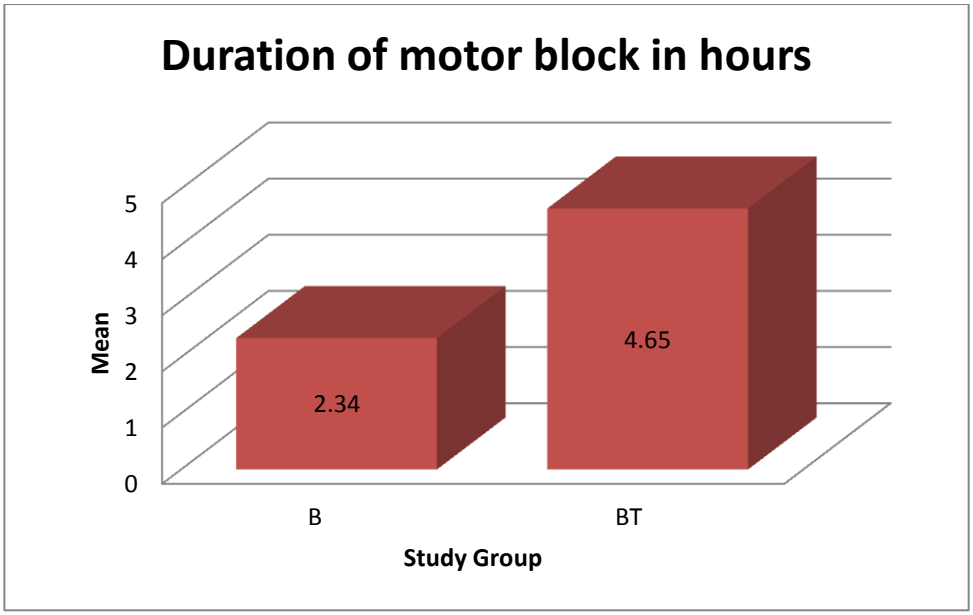
5. Duration of motor block in hours:

The duration of motor blockade in Group B was 2.34 ± 0.362 hours and in Group BT, was 4.65 ± 0.654 hours as shown table 11,graph 10.The statistical analysis by students‘t’ test showed that the time for duration of motor blockade in group BT was significantly longer when compared to Group B (P < 0.05).

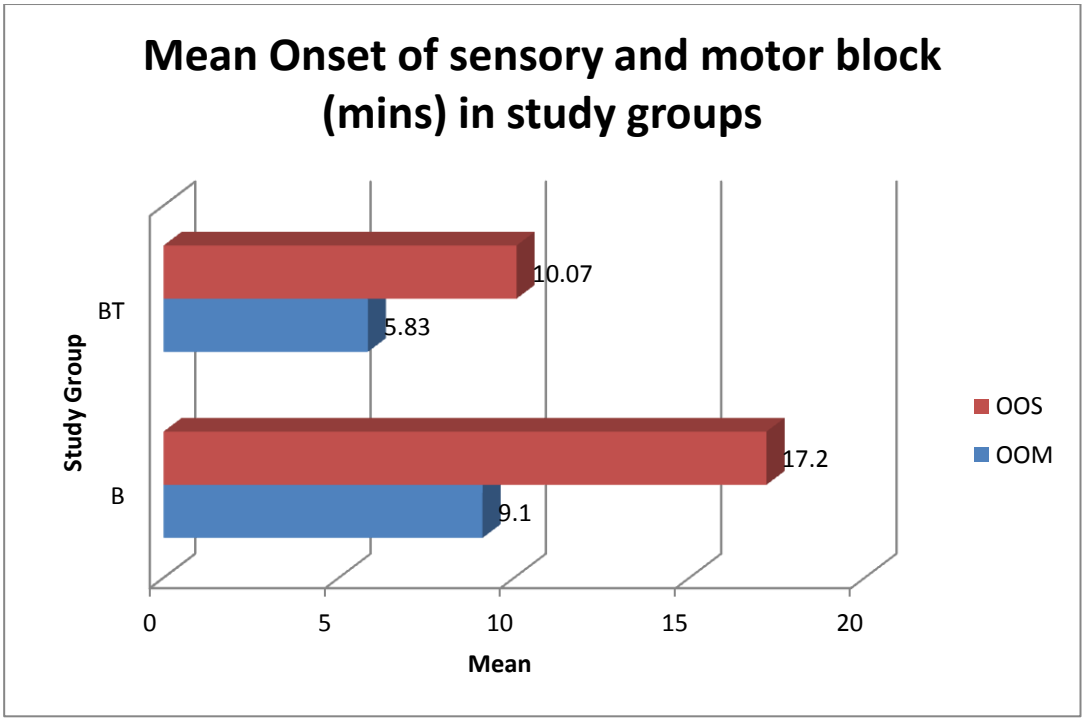
Table: 11

Duration of motor block in hours.	Mean	S.D	Statistical inference
B (n=30)	2.34	.362	T=-16.916 .000<0.05 Significant
BT (n=30)	4.65	.654	

Graph: 10

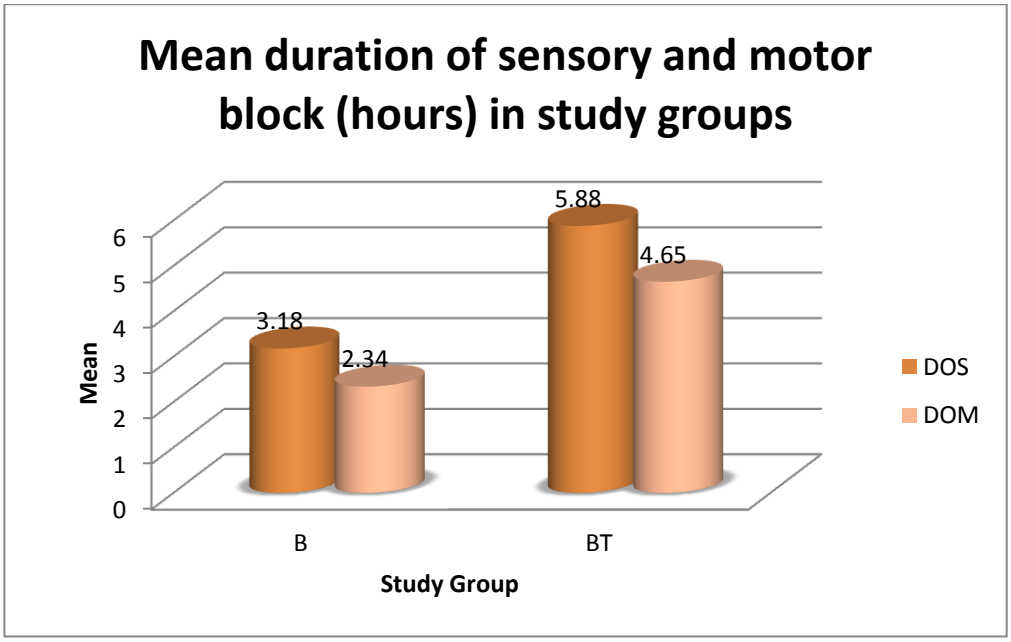


GRAPH 8:
MEAN ONSET OF MOTOR AND SENSORY BLOCK IN STUDY GROUPS



GRAPH 11:

MEAN DURATION OF SENSORY AND MOTOR BLOCK IN STUDY GROUPS



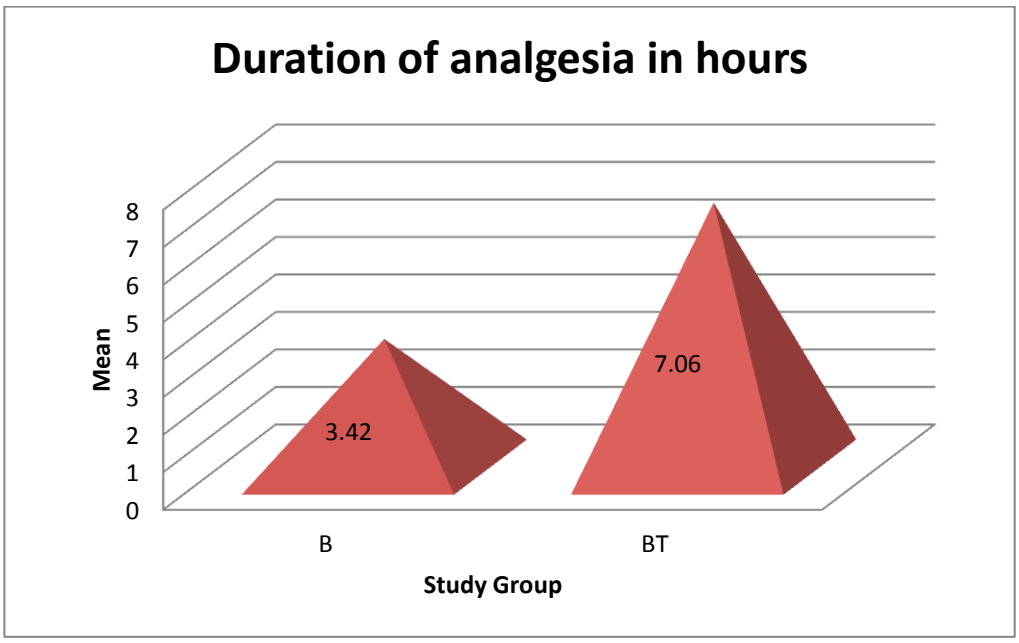
6.**Duration of analgesia in hours:**

The duration of analgesia in Group B was 3.42 ± 0.283 hours and in Group BT, was 7.06 ± 2.894 hours as shown table 12, graph 12.The statistical analysis by students 't' test showed that the time for duration of analgesia in group BT was significantly longer when compared to Group B ($p < 0.05$).

Table: 12

Duration of analgesia in hours	Mean	S.D	Statistical inference
B (n=30)	3.42	.283	T=-6.849 .000<0.05 Significant
BT (n=30)	7.06	2.894	

Graph: 12



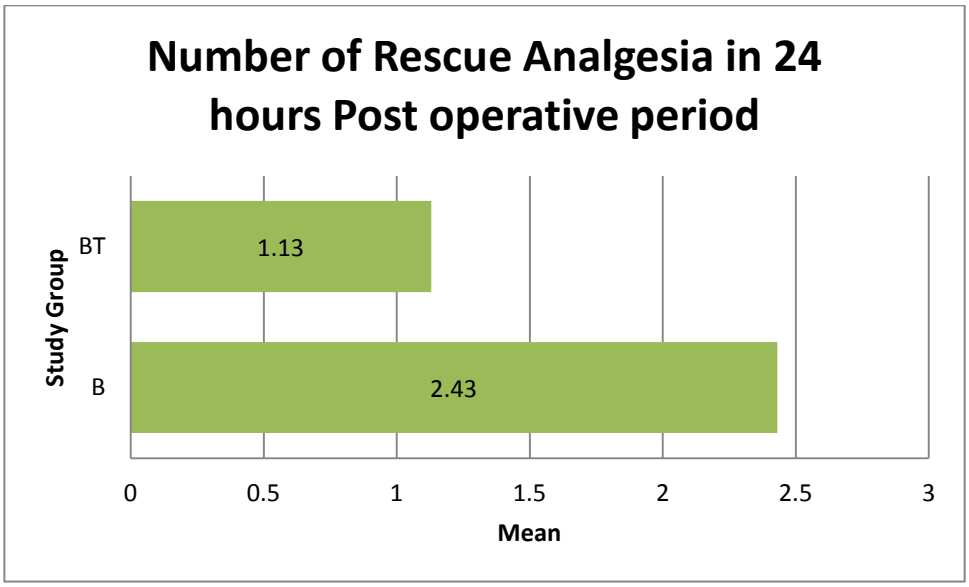
7. **Number of rescue analgesia in 24hours Post operative period**:

In Group B patients required 2.43± 0.568 rescue analgesic dosage and in group BT patients required only 1.13± 0.434 rescue analgesic doses in postoperative 24hours as shown in table 13 and graph 13.This difference in number of rescue analgesic doses required by patients of both groups is statistically significant ($p < 0.05$).

Table: 13

Number of rescue analgesics in 24hours Post operative period	Mean	S.D	Statistical inference
B (n=30)	2.43	.568	T=9.956 .000<0.05 Significant
BT (n=30)	1.13	.434	

Graph: 13



8. Hemodynamic variables: Pulse rate ,systolic blood pressure, diastolic blood pressure and oxygen saturation were recorded at 0min, 2 mins,5mins,10mins, 15mins,30 mins,1 hour, 2 hours, 3 hours,6 hours,12 hours and 24 hours.

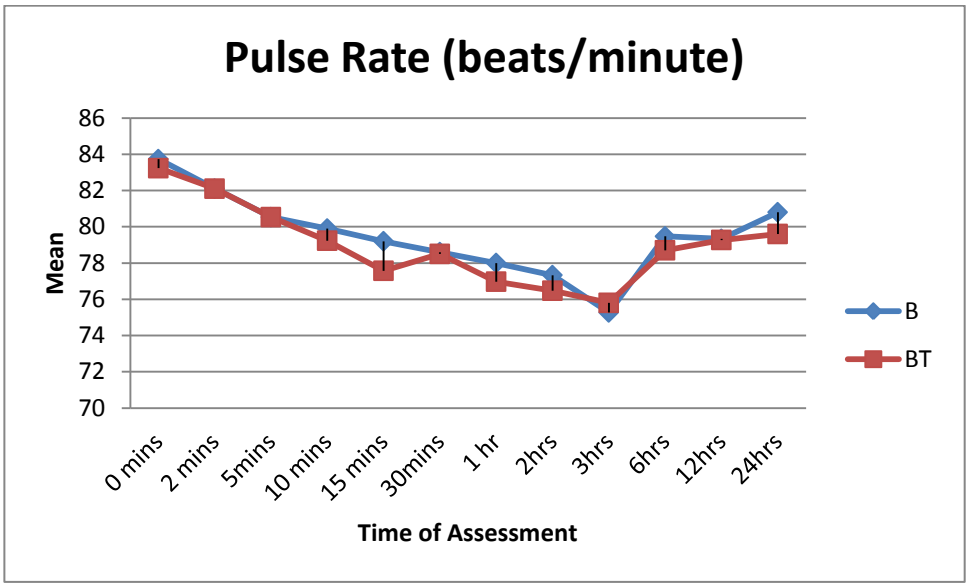
Pulse rate (beats/min)

There was no significant difference in pulse rate between the two groups as shown in table 14 and graph 14(p >0.05). None of the patients in both group developed bradycardia. Table: 14.

Pulse	Study group	Mean	S.D	Statistical inference
0minutes	B (n=30)	83.73	12.868	T=.152,0.880>0.05
	BT (n=30)	83.23	12.607	Not Significant
2minutes	B (n=30)	82.10	13.116	T=.000, 1.000>0.05
	BT (n=30)	82.10	12.115	Not Significant
5minutes	B (n=30)	80.53	12.294	T=.129, 0.898>0.05
	BT (n=30)	80.13	11.685	Not Significant
10minutes	B (n=30)	79.90	12.307	T=.217, 0.829>0.05
	BT (n=30)	79.23	11.482	Not Significant
15minutes	B (n=30)	79.20	11.883	T=.546, 0.587>0.05
	BT (n=30)	77.57	11.288	Not Significant
30minutes	B (n=30)	78.60	11.254	T=.035, 0.972>0.05
	BT (n=30)	78.50	10.919	Not Significant
1hour	B (n=30)	78.00	11.151	T=.368, 0.714>0.05
	BT (n=30)	76.97	10.604	Not Significant
2hour	B (n=30)	77.33	11.056	T=.312, 0.756>0.05
	BT (n=30)	76.47	10.421	Not Significant
3hours	B (n=30)	75.27	10.225	T=-.203, 0.839>0.05
	BT (n=30)	75.80	10.077	Not Significant
6hours	B (n=30)	79.47	11.301	T=.289, 0.774>0.05
	BT (n=30)	78.70	9.136	Not Significant
12hours	B (n=30)	79.33	10.337	T=.027, 0.978>0.05
	BT (n=30)	79.27	8.662	Not Significant
24hours	B (n=30)	80.80	10.317	T=.470, 0.640>0.05
	BT (n=30)	79.60	9.420	Not Significant

GRAPH 14:

CHANGES IN PULSE RATE (beats/minute)



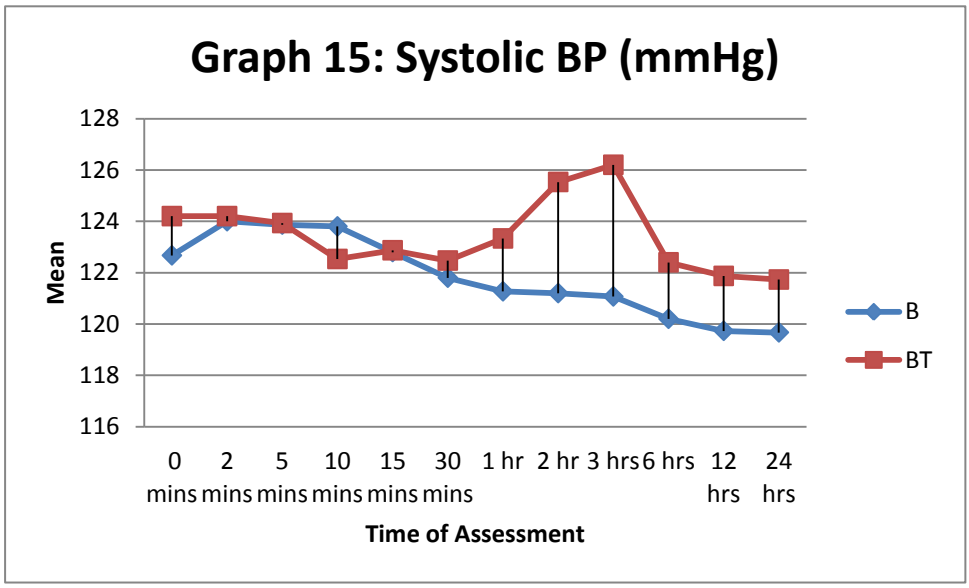
Systolic blood pressure (mmHg)

The mean systolic blood pressure between the two groups as shown in table 15 and graph 15 was comparable (p > 0.05).None of the patients in both group developed hypotension.Table:15

Time of Assessment	Groups	Mean	S.D	Statistical inference
0 mins	B (n=30)	122.67	9.444	T=-.647 .520>0.05 Not Significant
	BT (n=30)	124.20	8.919	
2 mins	B (n=30)	124.00	8.550	T=-.089 .930>0.05 Not Significant
	BT (n=30)	124.20	8.919	
5 mins	B (n=30)	123.87	8.629	T=-.030 .977>0.05 Not Significant
	BT (n=30)	123.93	8.843	
10 mins	B (n=30)	123.80	8.588	T=.569 .571>0.05 Not Significant
	BT (n=30)	122.53	8.645	
15 mins	B (n=30)	122.80	9.182	T=-.029 .977>0.05 Not Significant
	BT (n=30)	122.87	8.593	
30 mins	B (n=30)	121.80	8.684	T=-.285 .777>0.05 Not Significant
	BT (n=30)	122.47	9.449	
1 hr	B (n=30)	121.27	8.686	T=-.908 .367>0.05 Not Significant
	BT (n=30)	123.33	8.934	
2 hr	B (n=30)	121.20	8.704	T=-2.002 .052>0.05 Not Significant
	BT (n=30)	125.53	8.046	
3 hr	B (n=30)	121.07	8.610	T=-2.431 .081>0.05 Not Significant
	BT (n=30)	126.20	7.725	
6hr	B (n=30)	120.20	7.618	T=-1.152 .254>0.05 Not Significant
	BT (n=30)	122.40	7.171	
12hr	B (n=30)	119.73	7.196	T=-1.175 .245>0.05 Not Significant
	BT (n=30)	121.87	6.867	
24hr	B (n=30)	119.67	7.184	T=-1.171 .246>0.05 Not Significant
	BT (n=30)	121.73	6.470	

GRAPH 15:

CHANGES IN MEAN SYSTOLIC BLOOD PRESSURE (mmHg)



Diastolic blood pressure (mmHg)

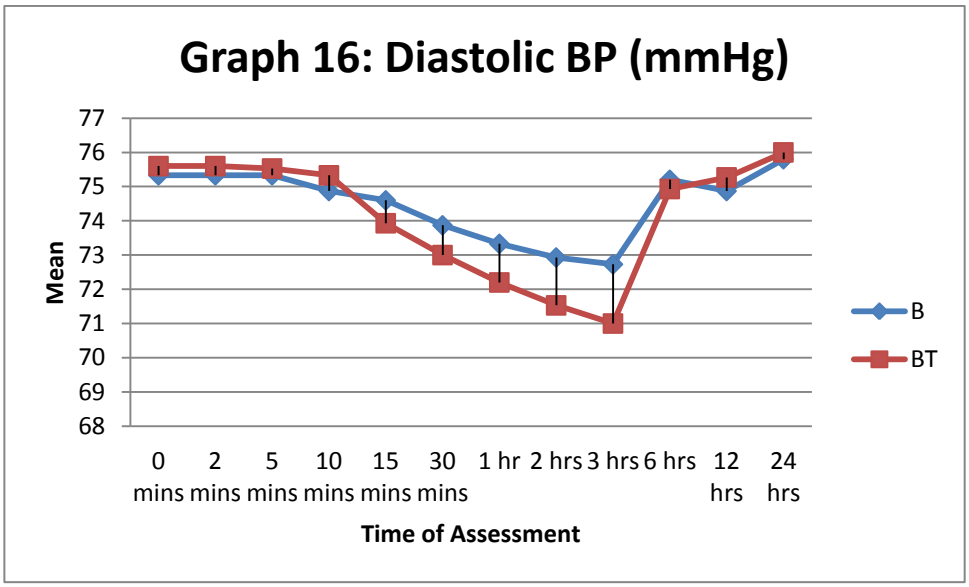
There was no significant difference in Diastolic Blood pressure between the two groups as shown in table 16 and graph 16 ($p > 0.05$)

Table: 16

Time of Assessment	Groups	Mean	S.D	Statistical inference
0 mins	B (n=30)	75.33	6.288	T=-.182 .856>0.05 Not Significant
	BT (n=30)	75.60	4.994	
2 mins	B (n=30)	75.33	6.288	T=-.182 .856>0.05 Not Significant
	BT (n=30)	75.60	4.994	
5 mins	B (n=30)	75.33	6.288	T=-.137 .892>0.05 Not Significant
	BT (n=30)	75.53	4.946	
10 mins	B (n=30)	74.87	5.794	T=-.340 .735>0.05 Not Significant
	BT (n=30)	75.33	4.795	
15 mins	B (n=30)	74.60	5.537	T=.490 .626>0.05 Not Significant
	BT (n=30)	73.93	4.996	
30 mins	B (n=30)	73.87	5.144	T=.674 .503>0.05 Not Significant
	BT (n=30)	73.00	4.807	
1 hr	B (n=30)	73.33	5.101	T=.899 .372>0.05 Not Significant
	BT (n=30)	72.20	4.649	
2 hrs	B (n=30)	72.93	4.891	T=1.142 .258>0.05 Not Significant
	BT (n=30)	71.53	4.599	
3 hrs	B (n=30)	72.73	4.968	T=1.381 .172>0.05 Not Significant
	BT (n=30)	71.00	4.749	
6 hrs	B (n=30)	75.20	4.944	T=.211 .834>0.05 Not Significant
	BT (n=30)	74.93	4.863	
12 hrs	B (n=30)	74.87	5.722	T=-.288 .775>0.05 Not Significant
	BT (n=30)	75.27	5.024	
24 hrs	B (n=30)	75.80	5.616	T=-.149 .882>0.05 Not Significant
	BT (n=30)	76.00	4.727	

GRAPH 16:

CHANGES IN MEAN DIASTOLIC BLOOD PRESSURE(mmHg)



SpO_2:

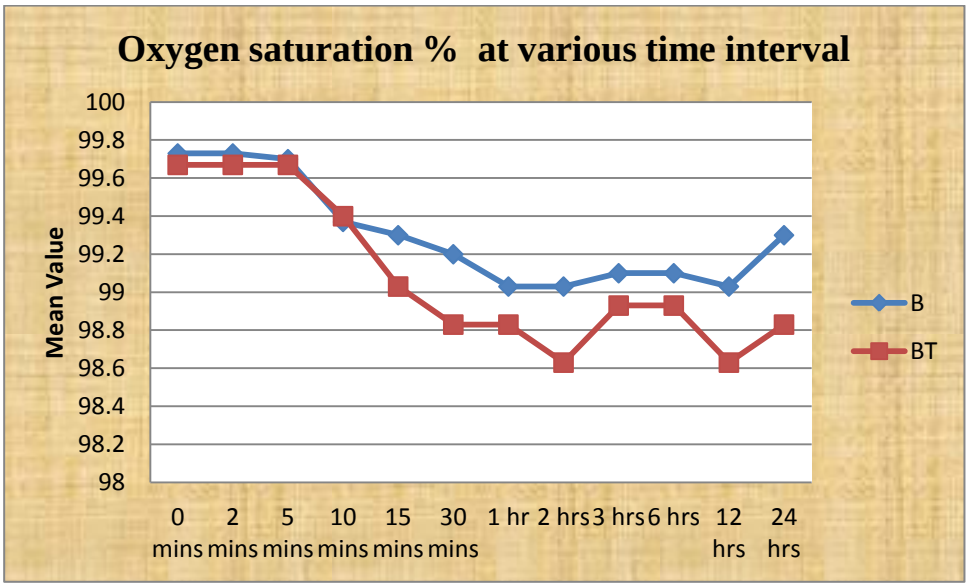
The statistical analysis by students 't' test showed that there was no significant difference in oxygen saturation between the two groups as shown in table 17 and graph 17 ($p > 0.05$)

Table: 17

Time of Assessment	Groups	Mean	S.D	Statistical inference
0 mins	B (n=30)	99.73	.521	T=.484 .630>0.05 Not Significant
	BT (n=30)	99.67	.547	
2 mins	B (n=30)	99.73	.521	T=.484 .630>0.05 Not Significant
	BT (n=30)	99.67	.547	
5 mins	B (n=30)	99.70	.535	T=.239 .812>0.05 Not Significant
	BT (n=30)	99.67	.547	
10 mins	B (n=30)	99.37	.556	T=-.209 .835>0.05 Not Significant
	BT (n=30)	99.40	.675	
15 mins	B (n=30)	99.30	.596	T=1.706 .093>0.05 Not Significant
	BT (n=30)	99.03	.615	
30 mins	B (n=30)	99.20	.610	T=1.257 .082>0.05 Not Significant
	BT (n=30)	98.83	.648	
1 hour	B (n=30)	99.03	.615	T=1.227 .225>0.05 Not Significant
	BT (n=30)	98.83	.648	
2 hours	B (n=30)	99.03	.669	T=1.412 .091>0.05 Not Significant
	BT (n=30)	98.63	.615	
3 hours	B (n=30)	99.10	.548	T=1.288 .203>0.05 Not Significant
	BT (n=30)	98.93	.450	
6 hours	B (n=30)	99.10	.548	T=1.288 .203>0.05 Not Significant
	BT (n=30)	98.93	.450	
12 hours	B (n=30)	99.03	.669	T=1.412 .091>0.05 Not Significant
	BT (n=30)	98.63	.615	
24 hours	B (n=30)	99.30	.596	T=1.904 .051>0.05 Not Significant
	BT (n=30)	98.83	.648	

GRAPH 17:

OXYGEN SATURATION



Comparison of side effects:

None of the patients in both the groups developed any complications.

Table : 18

Side effects	Group B	Group BT
Bradycardia	Nil	Nil
Hypotension	Nil	Nil
Nausea	Nil	Nil
Vomiting	Nil	nil

12. DISCUSSION

The supraclavicular brachial plexus approach is a very popular mode of anaesthesia, in which a small volume of solution can be delivered at a point where three trunks are compactly arranged, resulting in rapid onset of reliable blockade of the brachial plexus, to provide excellent anaesthesia for elbow, forearm and hand surgery and also provides good postoperative analgesia of short duration, even when a long acting local anaesthetic like bupivacaine is used alone. The nerve stimulator can be used to aid the location of the brachial plexus and plain bupivacaine used by this method has been claimed to produce the block as long as 3 – 8 hours. Practically the same result couldnot be produced in series of study with sole bupivacaine. To extend the analgesia beyond the operation rooms, various local anaesthetic action like continuous infusion of local anaesthetic via in dwelling catheters, use of different additives in local anaesthetics like narcotics, opioids, calcium channel blockers and benzodiazepine have been added to the local anaesthetics and their effect on the quality of block studied. A variety of opioids have been studied for brachial plexus blockade including tramadol hydrochloride.

Tramadol is known to produce antinociception and to enhance the effect of local anaesthetic. Tramadol produces this effect by its dual mechanism of action. Firstly it stimulates μ receptor and to lesser extent δ and κ - opioid receptors. Secondly it activates spinal inhibition of pain by decreasing the reuptake of norepinephrine and serotonin (non opioid mechanism) in peripheral nerve blocks. Several studies have demonstrated the advantage of using tramadol hydrochloride through various routes for analgesia.

Hence an attempt has been made to assess the efficacy of tramadol (2mg/kg) as an adjuvant to bupivacaine (0.25%) in brachial plexus block (supraclavicular approach) in terms of onset time, duration of analgesia, hemodynamic variables and rescue analgesic requirements in the first 24 hours.

We used nerve stimulator technique which has the advantage of minimizing neuropathy by avoiding actual physical contact with a nerve compare to paresthesia technique. When an electrical current is used to stimulate a nerve, at lower current the motor fibres depolarizes than the sensory fibers leading to a painless visible muscle contraction without eliciting a paresthesia. The high success

rate and absence of complications in performing the subclavian perivascular technique of brachial plexus block by nerve stimulator indicate that our technique is safe and effective also said by Carlo D. Franco et al[35] in his study.

A volume of 40ml of local anaesthetic agent was taken as this volume was associated with a more complete spread for brachial plexus block as found by Winnie and colleagues[36].

The particular dose of Tramadol 2mg/kg (100mg) was selected after previous studies like Kapral et al[24], Antonucci et al[19], Renu Wakhlo et al[18], Geze et al[20] and Siddiqui AS et al[28] used the same dosage in peripheral nerve block without any significant adverse effects.

A total of 60 patients within the age group of 19-72 were in included in the study, 30 in each Group B and Group BT.

Onset of Action:

In our study we found that the onset of sensory and motor block were significantly faster in patients who received a combination of tramadol and bupivacaine. Onset of motor block (group BT, 5.83 ± 1.053 min; group B, 9.10 ± 1.373 min).Onset of

sensory block (group BT 10.07 ± 1.837 min; group B 17.20 ± 2.140 min).

This could be due to a local direct action of Tramadol and its synergistic action with that of local anaesthetics. The onset of motor block was significantly faster than the onset of sensory block in both groups, this can be explained by 'Core and Mantle' concept of Winnie et al 1977[2], He observed and attributed this to the somatotrophic arrangement of fibres in a nerve bundle at the level of the trunks in which motor fibres are located more peripherally form the mantle and are blocked earlier than the sensory fibres at the core. Hence a local anaesthetic injected perineurally will begin to block the motor fibres before it arrives at the centrally located sensory fibres.

Duration of Motor and Sensory Block:

In our study mean duration of motor block was prolonged when tramadol was added to bupivacaine. (Group BT, 4.65 ± 0.654 hours; Group B, 2.34 ± 0.362 hours). In our study, the mean duration of sensory block was significantly higher ($P < 0.05$) in group BT than in group B. (Group BT, 5.88 ± 0.669 hours; Group B, 3.18 ± 0.524 hours).

Our results showed that sensory block tended to last longer as compared to motor block which agrees with the observation by de Jong et al[37]. These authors explained that large fibres require a higher concentration of local anaesthetic than small fibres. The minimal effective concentration of local anaesthetic for large (motor) fibres is greater than for small (sensory) fibres.Thus, motor function returns before pain perception and duration of motor block is shorter than the sensory block.

Duration of Analgesia:

In our study duration of analgesia (from onset of blockade to requirement of first supplement analgesic) was significantly higher in Tramadol Group BT (7.06 ± 2.894) compared to Group B (3.42 ± 0.283).

These results are comparable with the study of Suman Chattopadhyay et al[17].

Tramadol as analgesic adjuvants:

Various studies of Tramadol used in peripheral nerve block showed that Tramadol with Bupivacaine improves analgesic characteristics compared to Bupivacaine alone when administered for various peripheral nerve blocks.

Renu Wakhol et al[18] showed addition of (100 mg) 2 mg/kg of Tramadol to local anesthetic was found to be good agent for hastening the onset and prolonging sensory and motor block.

Sebastein Robaux et al[22] found that addition of tramadol to local anaesthetic agents improved the onset and duration of motor blockade. Antonucci S et al[19] found that Tramadol 100mg useful alternative, as adjuvant in peripheral block with lower incidence of side effects.

W. Kunapis et al[27] showed that adding Tramadol to Bupivacaine for brachial plexus block provides faster onset and longer duration of analgesia, improves the quality of analgesia. Siddiqui AK et al[28] studied addition of Tramadol 1mg/kg (50 mg) and (100mg) 2 mg/kg. Suggested that Tramadol 100mg is beneficial additive to lignocaine for IVRA since it shortened the onset of sensory block, enhanced the tourniquet tolerance and improved the perioperative analgesia.

All the studies are comparable with our results.

Tramadol has a local anaesthetic effect on peripheral nerves as this could provide potentially a synergistic effect in continuous

infusion as an additive to local anaesthetic agent has been studied by J.Balavenkatasubramanian[34].

Rescue Analgesia:

In our study, the number of patients who required rescue analgesia was also significantly lower in patients in Group BT. Similar observation was made in the above mentioned study by Suman Chattopadhyay et al[17]. The prolonged analgesia in Group BT could be due to local anaesthetic type effect of Tramadol on peripheral nerves as demonstrated by Yu-Chan Tsai et al[31].

Tramadol, an analgesic with peripheral effects similar to clonidine, moderately increases sensory block duration when compared with placebo or systemic control as mentioned in study by Joseph M. Neal et al[38]. Adding small doses of opioids to local anaesthetic solutions for peripheral blocks have resulted in improvement in the onset time, quality and duration of nerve block.

Side effects:

No significant side effects like respiratory depression, pneumothorax, signs and symptoms of local anaesthetic toxcity or neurological sequale were observed in any of the two groups. The lack of significant side effects like respiratory depression and

sedation make Tramadol as an adjuvant for supraclavicular brachial plexus block.

Haemodynamic parameters

In this study there was no significant change in the haemodynamic parameters between the groups. This was consistent with the observation by Suman Chattopadhyay et al[17].

In conclusion, Tramadol 100mg (2 mg/kg) when added to 38mL of Bupivacaine 0.25% for supraclavicular brachial plexus block speeds the onset of sensory and motor blocks ($P < 0.05$). The combination produces improved analgesia, resulting in a prolonged effect and reduced requirements for rescue analgesics.

13. SUMMARY

We conducted this study at Thanjavur Medical College and Hospital in 60 patients of both sex in age group of 19 to 72 years belonging to ASA I and II and their weight ranging in between 55 to 80years posted for various upper limb surgeries under subclavian perivascular approach of brachial plexus block with nerve locator.

The patients in group B received 38ml of 0.25% Bupivacaine and 2 ml Normal saline. In group BT received 38ml of 0.25% Bupivacaine and 2ml (2mg/kg) Tramadol.

Parameters observed were time of onset of sensory block and motor block, duration of motor blockade, and sensory blockade, duration of analgesia, sedation score and side effects.

This study shows that

Addition of tramadol to bupivacaine, when compared to bupivacaine alone, shows

1. Earlier the onset of motor and sensory blockade
2. increases the duration of motor and sensory blockade

3. significantly prolongs the duration of analgesia
4. Requirement of rescue analgesic in postoperative period 24 hours is less
5. Does not cause significant haemodynamic changes, respiratory depression, sedation or other adverse effects.

14. CONCLUSION

From our study we conclude that the addition of the tramadol 2mg/kg to 0.25% bupivacaine solution in brachial plexus block shows early onset of sensory and motor blockade and prolongs the duration of analgesia when compared to Bupivacaine alone. There are no significant side effects like respiratory depression and sedation. Hence tramadol may be considered as a useful adjuvant for bupivacaine when used for brachial plexus block.

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PROFORMA

A STUDY OF THE EFFICACY OF TRAMADOL AS AN ADJUVANT TO BUPIVACAINE IN BRACHIAL PLEXUS BLOCK

Name: I.P. No. :

Age: Department:

Sex: Date:

Preoperative Observations:

General Physical Examination:

Pulse rate: Blood Pressure:

Respiratory rate: Weight:

Systemic Examination:

C.V.S:

R.S: Others :

Investigations:

Hb%: R.B.S: ECG:

Blood Urea: S.Creatinine: Urine routine:

Preoperative Diagnosis:

Proposed surgery:

ASA Grade: 1 / 2

STUDY PROTOCOL:

Drug and Dosage Group: B: 38ml of Bupivacaine 0.25% + 2ml NS

BT: 38ml of Bupivacaine 0.25% + 2ml Tramadol (2mg/kg)

Observations:

1) Time of Injection: 0 min

2) Time of onset of sensory blockade: _____min

3) Time of onset of motor blockade: _____min

4) Quality of analgesia: Complete / Partial / Nil

MONITORING:

Time	**Pulse rate Per min**	**Systolic BP mmHg**	**Diastolic BP mmHg**	**Spo2 %**
0				
2				
5				
10				
15				
30				
1hr				
2hr				
3hr				
6hr				
12hr				
24hr				

5) Duration of surgery: _____ mins, _____ hours

6) Duration of sensory blockade: _____mins, _____ hours

7) Duration of motor blockade: _____ mins,_____hours

8) Duration of analgesia: _____ mins,_____hours

9) No. of rescue analgesics in post-op 24 hours: _____

Untoward effects, if any:

Supplementation, if any:

GROUP B - BUPIVACAINE

SL. No	Age	Sex	Wt in Kg	ASA Status	Diagnosis & Procedure	onset of Sensory block in Mins.	onset of Motor block in Mins	Duration of sensory block in Hrs.	duration of Motor block in Hrs.	Duration of Analgesia in hrs	Duration of surgery in Hrs	Side effects	No. of RA IN 24hrs Post op
1	66	M	70	II	Dupuytr. Contrac.-Rt relese	16	8	3.6	2.8	3.5	1	nil	2
2	48	M	73	I	Nec. fascitis Rt arm -W Debd.	18	11	3.8	2.6	4	1.5	nil	2
3	28	M	72	I	Lt Radius #- ORIF	16	7	2.7	2.3	3.2	1.6	nil	3
4	37	M	63	I	Lt Olecranon #-ORIF	15	8	2.5	2	3.6	2	nil	2
5	50	F	65	I	Rt Thumb injury-Repair	20	10	3.4	2.2	3.4	1.5	nil	2
6	72	M	65	II	BB# Rt FA- ORIF	18	10	2.2	2	3.5	2	nil	2
7	24	M	63	I	Cut injury Lt hand- Repair	18	10	3.8	3	3.3	1.5	nil	3
8	50	F	68	I	Cut injury Lt hand- Repair	20	9	3.6	2.4	3	1.5	nil	2
9	24	F	63	I	Rt Radius head #-Excision	12	9	3	2	2.9	1.5	nil	3
10	30	F	64	I	Tendon injury R-Hand -repair	21	8	3	2.1	3.5	2.2	nil	2
11	26	F	65	I	PIP Jt. dislocation F3 K-wire FIX	17	8	2.5	2	3.5	1	nil	2
12	27	M	66	I	PBC-Lt hand-Release &SSG	14	9	3.3	2.4	3.5	2.5	nil	2
13	35	F	60	I	PBC LT Hand_Release@SSG	18	9	2.7	2	3	2.5	nil	3
14	40	M	68	I	Flap cover Rt FA-Flap thining	18	11	4.1	2.8	3.6	2	nil	1
15	19	M	70	I	RawareaLt Thumb_SSG	19	7	2.8	2.3	3.2	1.5	nil	3
16	38	M	63	I	Lt claw hand-TendonTransfer	20	8	3.2	2.5	3.5	3	nil	3
17	37	M	76	I	Cut Injury Rt hand-Repair	17	9	3.1	2.4	3.5	1.5	nil	2
18	48	M	74	I	BB#F Arm Lt_ORIF	16	10	2.8	2	3.4	1.6	nil	3
19	34	M	76	I	EXT TEN injuryLt hand_Repair	17	10	3	3.2	3.6	2	nil	3
20	21	F	57	I	Flex Ten InjuryRt FA-Repair	18	14	2.8	2	3	2	nil	3
21	37	M	80	I	Cut InjuryRt F2,3,4_Repair	16	8	4	2	3.5	2	nil	3
22	38	M	62	I	RawareaLt FArm_SSG	17	9	3	1.8	4	2.5	nil	2
23	45	F	64	II	PBC RT Hand_Release@SSG	15	10	3.1	2.2	3.2	2	nil	3
24	28	F	66	I	BB# Lt FA-ORIF	19	9	3.4	2.4	3.5	3	nil	2
25	28	M	71	I	#Radius head Lt_Excision	15	8	3.1	2.3	3.1	2	nil	2
26	44	M	74	I	Cut InjuryLt Hand_REPAIR	17	9	3.2	2.4	3.3	1.5	nil	3
27	51	F	59	II	#Olecranan Lt-ORIF	16	8	3.5	2.8	3.6	2	nil	2
28	26	F	55	I	#SOH Rt_ORIF	14	9	2.2	1.8	3.2	2	nil	2
29	49	F	64	I	Colles#Rt FA_ORIF	21	9	3.8	2.6	4	1.8	nil	3
30	65	F	65	II	#BB Lt FA_ORIF	18	9	4.1	2.8	3.5	2	nil	3

GROUP-B - BUPIVACAINE PULSE RATE CHART (beats/mins)

S.No	GROUP	0 Mins.	2 Mins	5Mins	10 Mins	15 Mins	30 Mins	1hrs	2hrs	3hrs	6hrs	12hrs	24hrs
1	B	90	94	93	92	90	85	85	84	84	90	85	88
2	B	110	112	110	110	108	107	106	104	98	98	96	110
3	B	78	75	74	74	72	72	72	72	70	80	75	78
4	B	84	84	82	80	78	77	76	74	74	76	80	80
5	B	120	116	108	106	104	101	100	99	98	100	100	96
6	B	114	108	104	104	102	100	98	98	97	97	96	98
7	B	82	78	76	77	77	77	77	76	76	78	77	77
8	B	72	66	64	64	64	64	65	64	64	72	66	68
9	B	84	80	78	76	76	76	75	74	72	78	80	80
10	B	77	74	72	72	72	72	70	70	70	77	70	70
11	B	73	68	66	66	66	66	66	65	64	64	65	68
12	B	88	84	82	82	80	80	80	78	76	70	80	78
13	B	78	72	72	71	70	70	72	70	70	70	74	75
14	B	79	74	73	73	72	72	70	70	68	76	71	70
15	B	77	75	74	74	74	74	74	72	70	70	74	78
16	B	81	80	78	77	77	77	76	76	74	77	79	81
17	B	86	87	85	84	84	84	82	82	80	85	85	80
18	B	78	80	78	75	74	74	72	72	70	90	75	75
19	B	72	70	68	65	65	64	64	66	64	65	74	69
20	B	74	77	72	70	70	70	68	66	65	70	70	78
21	B	76	74	75	76	75	75	75	73	71	71	71	78
22	B	77	76	75	75	76	76	75	75	71	75	70	75
23	B	71	70	70	69	68	69	66	67	65	66	71	77
24	B	79	77	77	78	76	76	75	75	73	75	80	75
25	B	69	71	71	70	70	69	71	70	68	70	70	74
26	B	93	93	91	91	91	89	89	89	84	100	102	91
27	B	89	88	88	88	88	87	87	87	83	81	82	94
28	B	92	92	92	90	90	90	90	89	84	91	93	93
29	B	70	69	70	70	70	69	68	67	65	71	74	74
30	B	99	99	98	98	97	96	96	96	90	101	95	96

GROUP B BUPIVACAINE SYSTOLIC BLOOD PRESSURE CHART(mmHg)

S.No	0 Mins	2 Mins	5Mins	10Mins	15Mins	30Mins	1hr	2hr	3hr	6hr	12hr	24hr
1	110	120	118	118	110	110	110	110	110	110	110	110
2	140	140	140	140	140	140	140	140	140	140	140	140
3	130	130	130	130	130	130	128	128	128	130	130	130
4	130	130	130	130	130	130	130	130	130	130	130	130
5	130	130	130	130	130	128	128	128	128	120	120	120
6	120	120	120	120	120	120	120	118	118	120	120	120
7	120	120	120	120	120	120	120	120	120	120	120	120
8	120	120	120	120	120	116	116	116	116	116	120	120
9	120	130	130	130	120	120	118	118	118	120	120	120
10	130	130	130	130	130	128	128	128	128	128	126	120
11	130	130	130	130	130	128	128	128	128	128	120	120
12	120	120	120	120	120	120	118	118	118	120	120	120
13	120	120	120	120	120	120	118	118	118	118	120	120
14	110	110	110	110	110	110	110	110	110	110	110	110
15	130	130	130	130	130	130	128	128	126	120	120	120
16	110	120	118	118	116	110	110	110	110	110	110	110
17	110	110	110	110	110	110	110	110	110	110	110	110
18	130	130	130	128	128	126	126	126	126	126	120	120
19	120	120	120	120	120	120	118	118	118	120	118	120
20	110	110	110	110	110	110	110	110	110	110	110	110
21	130	130	130	130	130	128	128	128	128	120	120	120
22	110	120	120	120	110	110	110	110	110	110	110	110
23	120	120	120	120	120	120	120	120	120	120	120	120
24	120	120	120	120	120	120	120	120	120	120	120	120
25	140	140	140	140	140	136	136	136	136	130	128	130
26	110	110	110	110	110	110	110	110	110	110	110	110
27	120	120	120	120	120	120	118	118	118	120	120	120
28	140	140	140	140	140	136	136	136	136	130	130	130
29	120	120	120	120	120	120	118	118	118	120	120	120
30	130	130	130	130	130	128	128	128	126	120	120	120

GROUP B BUPIVACAINE DIASTOLIC BLOOD PRESSURES CHART (mmHg)

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hr	12hr	24hr
1	70	70	70	70	70	70	70	70	70	70	70	70
2	80	80	80	78	78	76	76	76	76	78	76	80
3	80	80	80	80	78	76	74	74	74	80	80	80
4	80	80	80	80	80	80	80	80	80	78	72	80
5	80	80	80	80	80	80	80	78	78	80	80	80
6	80	80	80	80	80	80	80	78	78	80	78	78
7	70	70	70	70	70	70	70	70	70	72	70	76
8	80	80	80	78	76	74	74	74	74	76	80	80
9	70	70	70	70	70	70	70	70	70	74	70	70
10	80	80	80	76	76	74	74	72	72	76	76	80
11	80	80	80	80	80	80	80	80	80	80	78	80
12	80	80	80	80	80	80	80	80	80	78	80	80
13	70	70	70	70	70	70	70	70	70	74	70	72
14	60	60	60	60	60	60	60	60	60	64	62	62
15	80	80	80	78	78	78	76	76	76	80	80	80
16	80	80	80	80	78	78	76	76	76	80	80	80
17	70	70	70	70	70	68	64	64	64	68	70	70
18	90	90	90	86	84	80	80	80	80	88	88	88
19	70	70	70	70	70	70	70	70	70	72	70	70
20	70	70	70	70	70	70	70	70	70	72	70	70
21	70	70	70	70	70	70	70	70	70	70	70	70
22	70	70	70	70	70	70	70	70	70	72	70	70
23	70	70	70	70	70	70	70	70	70	70	72	74
24	70	70	70	70	70	70	70	70	70	70	70	70
25	80	80	80	80	80	78	78	78	78	76	78	80
26	70	70	70	70	70	70	70	70	70	72	70	74
27	80	80	80	80	80	74	72	70	70	76	84	80
28	80	80	80	80	80	80	78	74	70	78	80	80
29	70	70	70	70	70	70	70	70	68	72	72	70
30	80	80	80	80	80	80	78	78	78	80	80	80

GROUP B - BUPIVACAINE SPO2% CHART

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hr	12hr	24hr
1	100	100	100	100	100	100	100	100	99	99	100	100
2	100	100	100	100	100	99	99	99	99	99	99	100
3	100	100	100	100	100	100	99	99	99	99	99	100
4	100	100	100	100	100	100	100	100	100	100	100	100
5	100	100	99	98	98	98	98	98	98	98	98	98
6	99	99	99	99	99	99	99	99	99	99	99	99
7	100	100	100	99	99	99	99	99	99	99	99	99
8	99	99	99	99	99	99	99	99	99	99	99	99
9	100	100	100	99	99	99	99	99	99	99	99	99
10	100	100	100	100	100	99	99	98	99	99	98	100
11	100	100	100	99	99	99	99	99	99	99	99	99
12	100	100	100	99	99	99	99	99	99	99	99	99
13	100	100	100	99	99	99	99	99	99	99	99	99
14	100	100	100	100	100	99	99	99	99	99	99	100
15	100	100	100	99	99	99	99	99	99	99	99	99
16	100	100	100	99	99	99	99	99	99	99	99	99
17	100	100	100	100	99	99	99	99	99	99	99	99
18	99	99	99	99	99	98	98	98	99	99	98	99
19	100	100	100	99	99	99	99	99	99	99	99	99
20	100	100	100	99	99	99	98	98	98	98	98	99
21	100	100	100	99	99	99	98	98	98	98	98	99
22	99	99	99	99	98	98	98	98	99	99	98	98
23	98	98	98	99	99	99	99	99	99	99	99	99
24	100	100	100	100	100	100	99	99	99	99	99	100
25	100	100	100	100	100	100	100	100	100	100	100	100
26	100	100	100	100	100	100	99	99	99	99	99	100
27	99	99	99	99	99	99	99	100	100	100	100	99
28	100	100	100	100	100	100	100	100	100	100	100	100
29	100	100	100	100	100	100	100	100	100	100	100	100
30	99	99	99	99	99	100	100	100	100	100	100	99

GROUP BT - BUPIVACAINE AND TRAMADOL

SL. No	Age	Sex	Wt inKg	ASA Status	Diagnosis & Procedure	onset of Sensory block in Mins.	onset of Motor block in Mins	Duration of sensory block in Hrs.	duration of Motor block in Hrs.	Duration of analgesia in hrs	Duration of Surgery in hrs	Side effects	No. of RA IN 24hrs Post op
1	28	M	62	I	#Lt Thumb ppx-ORIF	12	5	5	4.2	5.5	1.5	nil	2
2	50	F	65	I	BB#R F-arm_ORIF	11	7	6.3	5.4	7.3	2	nil	1
3	34	M	63	I	R-F3,Tendon injury_Repair	9	6	5.8	4.6	7	2.5	nil	1
4	40	M	66	I	Lt ElbowTumor_EX,BIOPSY	13	5	6	4.2	7.1	1	nil	1
5	28	M	64	I	Cut Injury R-wrist_Repair	9	4	5.7	4.7	6.5	2	nil	1
6	32	M	66	I	Degloving Injury R Hand_W.DEB	9	6	6.2	5.5	7	1.5	nil	1
7	30	F	60	I	BB#L F-arm_ORIF	11	7	6	5.4	6.5	2	nil	2
8	36	F	61	I	BB#R F-arm_ORIF	10	8	7.1	3.9	22	2	nil	0
9	28	M	67	I	FT InjuryR hand_Repair	6	4	6.8	5.2	7.5	2	nil	1
10	52	M	68	II	Galazzi#Lt hand_ORIF	12	6	7.6	3.8	6.5	2	nil	1
11	36	F	66	I	#SOH R _ORIF	12	5	6	4.8	7	2.5	nil	1
12	28	M	69	I	Cut Injury R-Hand_Repair	11	5	5.1	4.2	6.1	1.5	nil	1
13	36	M	68	I	#Distal ULNA_ORIF	10	7	5	5.5	6	2	nil	1
14	32	M	72	I	Trumatic Ampu RF_RevisionAmpu	10	7	5.5	5.2	6.2	1.5	nil	1
15	34	M	76	I	#SOH Lt_ORIF	11	7	6.7	4.5	7	2.5	nil	1
16	26	M	65	I	Galazzi#Rt hand_ORIF	9	6	6.1	5.4	6.2	2	nil	1
17	60	M	70	II	#SOH Lt_ORIF	6	5	5.5	4.8	7	2	nil	1
18	42	M	78	I	BB#L F-arm_ORIF	13	6	5	4.1	5	1.5	nil	2
19	55	M	65	II	#Radialhead Lt -Exision	9	6	6.2	3.4	6.5	1.5	nil	1
20	25	M	73	I	Gangrene Lt hand_Amputation	13	6	6	4.5	6.3	1.5	nil	1
21	55	M	68	II	#lt 2,3.ppx Lt hand_K-Wirefixation	9	7	6	4.9	6.5	1	nil	1
22	38	M	75	I	R-F3,#_K-Wire Fixation	10	5	5.2	5	6	1.5	nil	2
23	37	M	77	I	Rawarea Rt Hand_SSG	10	4	4.9	3.9	5.4	1.5	nil	1
24	19	M	63	I	PBC Rhand-Release@SSG	10	6	6.1	5.6	7.6	2	nil	1
25	27	M	65	I	PBC Lhand-Release@SSG	11	5	5	3.1	6.3	2	nil	1
26	55	F	62	II	Rawarea Rt F arm_SSG	12	6	6.2	4.8	7.3	1.5	nil	1
27	35	M	68	I	Rawarea Rt Hand_SSG	8	7	6.1	5.2	6.8	1.5	nil	1
28	20	M	64	I	Raw area Lt palm_SSG	8	7	6.1	5	7.5	1.5	nil	1
29	52	M	61	I	Lt BEStump_closure	10	5	6.1	4.5	6.1	1.5	nil	1
30	22	F	60	I	BB#Lt hand_ORIF	8	5	5	4.1	6	2	nil	2

GROUP BT - BUPIVACAINE AND TRAMADOL PULSE RATE CHART (beats/mins)

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hrs	12hrs	24hrs
1	76	75	76	77	75	78	76	79	80	72	79	80
2	82	81	80	84	81	83	90	84	86	80	86	81
3	90	90	82	80	84	84	83	82	84	81	83	83
4	110	106	102	100	98	98	96	95	94	98	90	92
5	82	82	80	80	76	76	75	75	74	80	82	85
6	88	86	85	85	84	84	82	82	82	85	82	88
7	72	72	70	70	68	68	66	65	65	70	72	65
8	84	84	80	80	76	75	74	74	72	80	76	80
9	77	76	75	74	72	72	74	74	68	74	76	74
10	73	72	70	68	66	68	67	67	66	65	66	70
11	88	86	85	85	84	84	82	82	82	85	82	80
12	78	75	74	72	70	70	69	69	68	70	75	76
13	120	118	115	114	110	110	106	105	100	94	99	100
14	114	112	110	106	105	104	100	100	98	110	101	98
15	79	78	75	74	72	74	70	70	70	76	72	73
16	77	76	75	74	72	76	72	70	70	76	70	75
17	81	80	78	76	75	77	76	76	76	80	80	82
18	86	85	84	83	80	82	80	78	78	80	82	82
19	78	76	75	75	72	76	74	74	74	74	72	76
20	72	72	70	68	68	69	70	70	68	72	78	68
21	74	74	72	70	70	70	68	68	68	75	66	62
22	76	75	74	72	70	70	66	66	66	70	78	76
23	77	76	75	74	72	74	72	70	70	78	72	80
24	71	70	68	68	66	66	66	66	66	70	70	69
25	79	78	76	75	74	74	72	72	72	74	79	70
26	69	68	66	65	64	66	64	62	62	72	78	90
27	93	92	90	90	88	88	85	85	84	80	88	88
28	89	88	86	85	84	86	84	84	84	82	90	85
29	92	90	88	86	85	86	84	84	82	81	85	91
30	70	70	68	67	66	67	66	66	65	77	69	69

GROUP BT BUPIVACAINE AND TRAMADOL
SYSTOLIC BLOOD PRESSURES CHART (mmHg)

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hrs	12hrs	24hrs
1	130	130	128	126	122	126	130	130	132	130	130	130
2	120	120	118	116	116	120	122	124	126	120	120	120
3	120	120	118	116	120	120	120	120	122	120	120	120
4	126	126	126	126	126	126	130	130	132	130	130	130
5	130	130	130	128	130	130	130	132	132	130	130	130
6	140	140	140	138	140	140	140	140	140	136	134	130
7	110	110	110	110	110	106	110	114	116	110	110	110
8	120	120	120	118	120	120	120	122	122	120	120	120
9	110	110	110	110	110	110	110	112	114	110	110	110
10	130	130	130	128	130	130	130	128	128	130	120	120
11	120	120	120	118	120	118	122	126	126	120	120	120
12	130	130	130	128	130	128	130	132	132	130	130	130
13	120	120	120	118	118	120	120	124	124	120	118	118
14	130	130	130	128	128	130	130	132	132	130	128	128
15	120	120	120	118	118	120	120	124	126	118	118	118
16	110	110	110	110	110	110	114	122	124	110	110	110
17	140	140	140	138	138	140	140	140	140	130	130	130
18	130	130	130	130	128	130	130	130	130	124	124	124
19	120	120	120	120	122	120	120	124	124	120	120	120
20	130	130	130	128	128	128	128	126	126	124	124	124
21	140	140	140	138	138	140	140	140	140	130	130	130
22	120	120	120	120	120	116	116	122	122	120	120	120
23	120	120	120	118	118	116	118	120	120	120	120	120
24	110	110	110	108	108	106	108	110	112	110	110	110
25	130	130	130	128	128	124	124	126	128	126	126	120
26	120	120	120	118	118	116	116	120	120	120	120	120
27	120	120	120	120	120	118	116	116	116	120	120	120
28	120	120	120	118	118	116	116	120	120	120	120	120
29	140	140	138	138	136	136	134	140	140	130	130	130
30	120	120	120	118	118	114	116	120	120	114	114	120

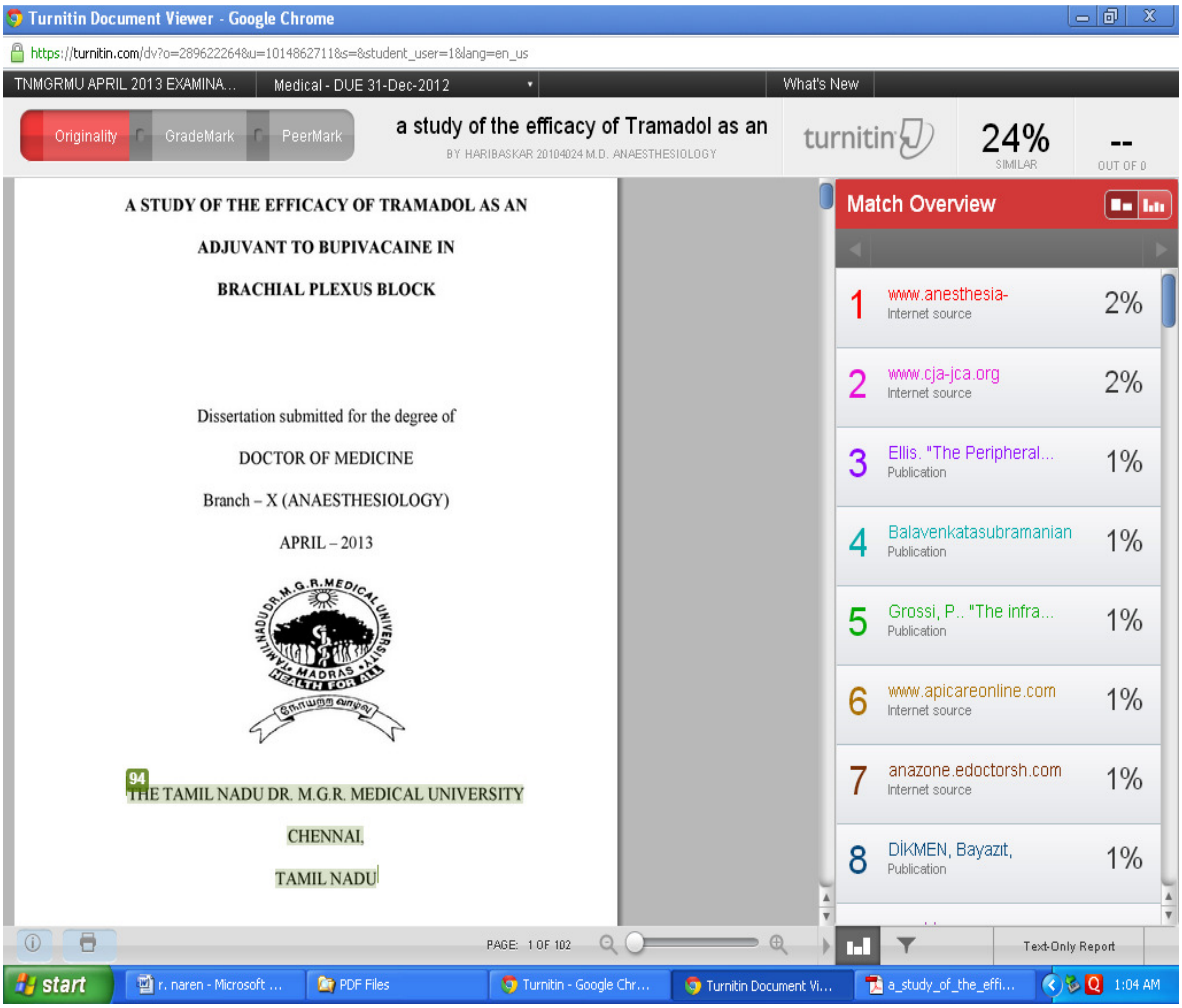
GROUP BT BUPIVACAINE AND TRAMADOL
DIASTOLIC BLOOD PRESSURES CHART (mmHg)

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hrs	12hrs	24hrs
1	80	80	80	78	78	76	76	76	76	76	80	76
2	80	80	78	78	76	76	74	74	72	74	80	80
3	70	70	70	70	68	64	64	64	64	70	70	70
4	78	78	78	78	76	70	70	70	70	70	70	70
5	80	80	80	80	76	76	74	74	74	80	80	80
6	80	80	80	80	80	76	76	74	74	80	80	80
7	70	70	70	70	70	70	68	68	68	70	70	74
8	70	70	70	70	68	68	68	68	66	70	70	74
9	80	80	80	80	78	78	76	76	74	80	80	80
10	80	80	80	80	78	78	76	76	76	80	80	80
11	70	70	70	70	70	70	70	70	70	70	70	72
12	80	80	80	80	78	78	78	76	74	80	80	80
13	80	80	80	80	78	76	76	74	74	80	80	82
14	70	70	70	70	68	66	66	66	66	70	70	70
15	70	70	70	70	68	68	68	66	64	70	70	70
16	70	70	70	70	68	68	66	66	64	70	70	72
17	80	80	80	80	78	78	78	78	78	78	78	80
18	80	80	80	80	78	78	76	76	74	80	80	80
19	80	80	80	80	80	78	78	76	76	80	80	80
20	70	70	70	70	68	68	68	66	66	70	70	72
21	80	80	80	80	80	78	78	76	76	80	80	80
22	80	80	80	80	80	78	76	76	76	80	80	80
23	70	70	70	70	68	68	68	68	68	70	70	72
24	70	70	70	70	68	68	66	64	64	70	70	72
25	80	80	80	80	78	78	76	74	74	80	80	84
26	70	70	70	70	68	68	66	66	64	70	70	70
27	70	70	70	70	70	70	70	70	70	70	70	70
28	70	70	70	70	68	68	68	66	66	70	70	70
29	80	80	80	78	78	78	76	76	76	80	80	80
30	80	80	80	78	78	76	76	76	76	80	80	80

GROUP BT - BUPIVACAINE AND TRAMADOL SPO2% CHART

S.No	0 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	1hr	2hrs	3hrs	6hrs	12hrs	24hrs
1	100	100	99	100	100	100	100	100	100	100	100	100
2	99	99	99	100	100	100	100	99	99	99	99	100
3	100	100	100	100	100	100	100	100	100	100	100	100
4	100	100	100	99	99	100	100	98	99	99	98	100
5	100	100	100	100	100	98	99	99	99	99	99	98
6	99	99	99	100	100	99	99	99	99	99	99	99
7	100	100	100	100	100	99	99	99	99	99	99	99
8	100	100	100	100	99	99	99	99	99	99	99	99
9	100	100	100	100	99	98	98	98	98	98	98	98
10	99	99	99	99	98	98	98	98	99	99	98	98
11	100	100	100	100	99	99	98	98	99	99	98	99
12	100	100	100	99	99	99	99	99	99	99	99	99
13	100	100	100	100	98	98	98	99	99	99	99	98
14	100	100	100	100	99	99	99	98	99	99	98	99
15	99	99	100	99	99	99	99	99	99	99	99	99
16	100	100	100	100	99	98	98	99	99	99	99	98
17	99	99	99	99	98	98	98	98	99	99	98	98
18	100	100	100	100	99	99	99	99	98	98	99	99
19	98	98	98	98	99	99	99	99	99	99	99	99
20	100	100	100	99	99	99	99	98	99	99	98	99
21	99	99	99	99	99	99	99	99	99	99	99	99
22	100	100	100	100	99	99	99	98	99	99	98	99
23	100	100	100	100	99	99	99	99	99	99	99	99
24	100	100	100	99	99	99	99	98	98	98	98	99
25	100	100	100	99	99	99	99	98	99	99	98	99
26	99	99	99	98	98	99	99	99	99	99	99	99
27	100	100	100	99	99	98	98	98	99	99	98	98
28	100	100	100	99	99	98	98	98	98	98	98	98
29	99	99	99	98	98	98	98	99	99	99	99	98
30	100	100	100	99	99	99	99	98	99	99	98	99

PLAGIARISM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